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DISEASES OF THE EYE AND EAR.

A MANUAL FOR STUDENTS AND PRACTITIONERS.

BY

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AUTHORS' PREFACE.

In the following pages, the authors have endeavored to present the subjects of Ophthalmology and Otology in as clear, thorough, and interesting a manner as the limited space would permit. Of necessity and intentionally, only the cardinal facts have been mentioned, since their experience as teachers has impressed the writers with the fact that a complex consideration is confusing to the beginner, but that after the fundamental principles have been mastered, the details of a more extensive work are readily acquired. While the matter has been prepared primarily for the use of the student, it is believed that it will prove of equal service to the practising physician in the management of his cases. To render the text more effective, numerous illustrations have been employed, which should be carefully studied in connection therewith. That the work might present a general résumé, the authors have consulted the principal authorities, to whom they acknowledge their indebtedness for many of the views contained herein.

New Haven, 1905. A. N. A.
Ann Arbor, 1905. O. A. G.

40126

EDITOR'S PREFACE.

IN arranging for the editorship of *The Medical Epitome Series* the publishers established a few simple conditions, namely, that the Series as a whole should embrace the entire realm of medicine; that the individual volumes should authoritatively cover their respective subjects in all essentials; and that the maximum amount of information, in letter-press and engravings, should be given for a minimum price. It was the belief of publishers and editor alike that brief works of high character would render valuable service not only to students, but also to practitioners who might wish to refresh or supplement their knowledge to date.

To the authors the editor extends his heartiest thanks for their excellent work. They have fully justified his choice in inviting them to undertake a kind of literary task which is always difficult—namely, the combination of brevity, clearness, and comprehensiveness. They have shown a consistent interest in the work and an earnest endeavor to coöperate with the editor throughout the undertaking. Joint effort of this sort ought to yield useful books, brief manuals as contradistinguished from mere compends.

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THE EYE AND ITS DISEASES.

CHAPTER I.

EXAMINATION OF THE EYE AND ITS APPENDAGES.

Lachrymal Apparatus.—Under favorable conditions the edge of the **lachrymal gland** may be felt as it lies in its fossa on the upper outer wall of the orbit, back of the orbital ridge. Enlargement, tumor, or prolapse should be made out. The presence of excess of tears in the conjunctival sac (epiphora, stillicidium) is indicated by a watery line along the edge of the lower lid and at the inner canthus. Attention should be turned to the **conducting apparatus**, and the small openings (puncta) on the margin of each lid near the inner canthus should be found open and lying against the eyeball. The region overlying the lachrymal sac is next examined for redness and swelling. The finger, with the ball turned toward the nose, is pressed firmly over the lachrymal sac, and at the same time the puncta are watched to observe the escape of discharge, which would indicate that the lachrymal (nasal) duct is closed. By introducing the point of a lachrymal syringe into the lower punctum, solutions may be forced into the lachrymal sac and out again through the upper punctum, if it seems desirable to determine the patency of these channels. The character of the fluid may also be observed with reference to the presence of abnormal secretion in the lachrymal sac.

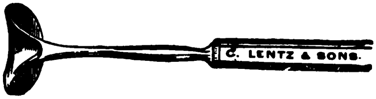
Lids.—The width and length of the opening between the upper and lower lids (**palpebral fissure**) should be observed, as well as any drooping of the upper lid (ptosis).

The thin **skin covering the eyelids** should then be examined for any disease which may occur there, and for œdema, swelling, or redness.

The **margins of the lids** should receive a thorough inspection. Note the number and character of the cilia, as well as their direction, being sure that no fine lashes are turned in against the eyeball (trichiasis), and that there is not a double row of lashes (distichiasis). Note the presence of redness, swelling, discharge, scales, crusts, watery cysts, ulcers, tumors, pediculi, or ova. If the upper or lower lid is found rolled inward, so that the margin is turned against the eyeball, the condition is called entropion. When the margin is turned outward, showing more or less of the conjunctiva, it is known as ectropion.

One should next investigate the **inner surface of the upper lid** by turning. This is accomplished by seizing the eyelashes with a firm hold between the thumb and finger of the left hand, with the thumb below. The patient must then look down, and any smooth instrument, preferably about the size of a match, should be pressed into the skin just under the edge of the orbital ridge. If this instrument is pressed down, folding the skin before it, while the eyelashes are pulled up outside the folded skin, the lid will turn, and may be held in place for inspection by the thumb, which is conveniently present. The inner surface of the lid should be examined as to the condition of the conjunctiva, congestion, thickening, granulations, or points of discoloration.

FIG. 1.



Desmarres' lid-retractor.

The **inner surface of the lower lid** may be examined by placing the finger well up to the edge and pulling down while the patient looks up.

To make satisfactory examination of the lids and eyeball in young children the head must be held face up between the surgeon's knees and the lids pried apart by the fingers or by the use of lid-retractors. (See Fig. 1.)

Conjunctiva.—The method of examining the **palpebral conjunctiva** has just been described. The transition of the palpebral into the ocular conjunctiva (**retrotarsal fold, fornix, cul-de-sac**) should not be overlooked. When the upper lid has been turned, the edge of the tarsal cartilage must then be raised by a blunt instrument.

The **ocular (bulbar) conjunctiva** is easily accessible, and congestion, thickening, œdema (chemosis), and tumors noted. By using pressure with the edge of the lid it will be seen that the conjunctiva is loosely attached to the eyeball, except about the cornea, where it is adherent by a narrow zone (limbus). Presence of lesions in this locality should be noted.

Conjunctival Discharge.—The student must learn to distinguish the various forms of discharge found in the conjunctival sac:

a. Watery (tears, epiphora).—Found in stenosis of conducting apparatus, etc.

b. Mucous.—Mucilaginous, but clear. Example, chronic conjunctivitis.

c. Mucopurulent.—Tenacious, white or yellow—as in acute and chronic conjunctivitis.

d. Purulent.—Creamy. Runs out of the eye when the lids are separated—as in gonorrhœal ophthalmia.

Congestion of the Eyeball.—*a. Conjunctival.*—This form may be easily distinguished by the fact that the vessels are movable with the conjunctiva over the eyeball. Found in conjunctivitis.

b. Ciliary or Circumcorneal.—A fine vessel congestion most intense about the periphery of the cornea. Pink or violaceous in color. Due to irritation or inflammation in cornea, iris, or ciliary body.

c. Scleral—Conjunctiva movable over it. May be *localized fine-vessel congestion* or *general* in form of large vessels which perforate the sclera. In scleritis or glaucoma.

Sclera.—The sclera may show congestion, localized swellings, bulging (staphyloma), or areas of discoloration due to scleritis or congenital.

Oblique Illumination.—Although the further examination is partly carried on by daylight, the method of oblique illumination in a dark room next comes into use. A double convex lens of about 2.5 inch focus is held so that the image of the artificial light (an Argand burner is best), which should be at least 2 feet away, is "played" upon the eye. The importance of this manœuvre is very great. Another lens may be used to magnify the illuminated field. Special instruments have been devised for this purpose, called corneal microscopes.

Cornea. The **anterior surface** of the cornea should be carefully examined by oblique illumination for irregularities, bloodvessels, foreign bodies, ulcers, blisters, depressions, and opacities. One may observe the reflection of a window when the patient is facing it, and note distortions in its outline by moving the eye.

The **deeper layers** of the cornea should be examined for opacities. A dense white opacity (leucoma), whether superficial or deep, or a moderately thick cloud (macula), may be easily discerned by daylight, but a faint opacity (nebula) is best seen by oblique illumination. The **posterior surface** should be scrutinized for opacities, usually punctate.

Sensibility of the cornea may be tested by brushing the surface with a wisp of cotton. Normally this is resented by a quick reflex.

Anterior Chamber.—The **depth** of the anterior chamber should be noted, i. e., the distance between the posterior surface of the cornea and the anterior surface of the iris and lens. The clearness of the **aqueous humor** should be noted as well as the presence of pus and exudate (hypopyon) or blood (hyphæma).

Iris. The **anterior surface** of the iris should be observed carefully and compared with the other eye. The muddy discoloration from congestion which is accompanied by loss of detail in the fine markings of this surface, as well as masses of exudate, tumors, or pigment-spots, will be recognized with a little experience. **Quivering of the iris** when the eye is moved (iridodonesis or tremulous iris) is sometimes seen when the lens is absent or dislocated.

Pupil.—The pupil should be circular and nearly in the centre of the iris. Great variation in size is possible under normal conditions. It is sometimes desirable to record its size, which may be done by comparing with an instrument which has numerous circular apertures of measured diameter (pupillometer). The **reaction of the pupil to light** may be roughly tested by alternately covering and uncovering the eyes with the hands before a window. A better way is to throw the light by oblique illumination in and out of the pupil in the dark room. The pupil into which the light is thrown should contract (direct action), but the other should do so as well (consensual action). When the patient looks from a distant to a near object, the pupil should also contract (**reaction to accommodation and convergence**).

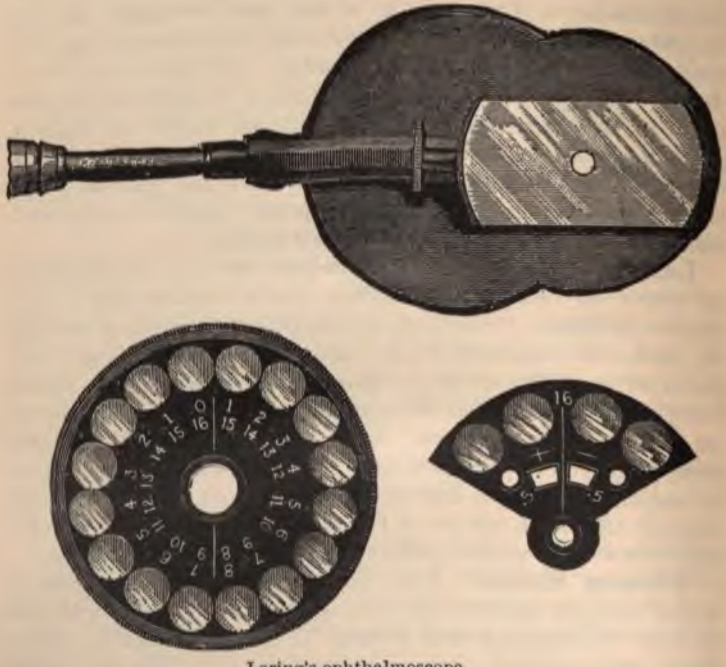
Lens.—The lens may be **examined** partially by daylight, or better by oblique illumination, as far as the size of the pupil will admit. The more complete examination as carried on with the ophthalmoscope will be described later. Its fixity of **position** should be determined. Dislocation (luxation) is evidenced by iridodonesis or by obtaining a view of its edge, which is never seen under normal conditions. **Opacities** (cataract) on the anterior or posterior surface (polar or capsular) or in the lens-substance are noted. A moderate amount of haze in advanced life, often quite brownish (sclerosis), is normally present and compatible with useful vision.

Vitreous Humor.—That part of the vitreous chamber which lies just back of the lens is accessible by daylight and oblique illumination, and should be perfectly clear. When filled with exudate, involved by a new growth or retinal detachment, these may be made out. The deeper parts of the vitreous are examined with the ophthalmoscope.

Orbit.—The finger should be passed about the bony edge of the orbit and pushed well back inside about the eyeball for the detection of tumors and irregularities. If orbital disease is suspected, the **nose** and **accessory sinuses** should be investigated.

Eyeball.—Note the **position** of the eyeball as to undue prominence (exophthalmos, proptosis) or recession into the orbit (enophthalmos), and as to whether it is pushed to one or the other side. Also whether the eyeball is larger (megalophthalmos) or smaller (microphthalmos) than the normal **size.**

FIG. 2.



Loring's ophthalmoscope.

Ophthalmoscope.—**Ophthalmoscopy** is a difficult art, and the beginner is advised to embrace every opportunity to perfect himself. The **principle** underlying the use of the ophthalmoscope should be thoroughly mastered. The reasons why the pupil appears dark and no view of the background dus) may be obtained under ordinary circumstances are two: first, because light which enters the eye is reflected back to its source; and secondly, because there is little internal reflection on account of the pigmented background. The problem of getting in the path of the light returning from the interior of the eye might be simply solved by holding a hollow tube in a candle-flame. Through this the pupil will appear luminous. Or, if a hole is made in the centre of a mirror and the light reflected into the pupil, the observer's eye, placed at this aperture, will see the fundus illuminated in the same way. This is essentially an ophthalmoscope.

The **modern complete** or **refracting ophthalmoscope** consists of a revolving disk, near the circumference of which is arranged a series of apertures filled with lenses of various strengths. These are made to pass back of the aperture in a concave mirror. Suitable support for the disk and mirror with handle make up the instrument.

There are **four methods of ophthalmoscopy for diagnosis of lesions:**

I. A **strong lens in the disk,** say +16 D., is turned behind the aperture. An Argand burner is placed on a level with the patient's eye, on the same side of the head, so that the shadow of the temple falls on the tip of the nose. The observer approaches the patient on the same side as the light, while reflecting it into the eye and looking through the aperture with the same eye as that observed. By this method the cornea, lens, and anterior part of the vitreous may be studied in great detail with magnified image.

II. With **no lens behind the aperture,** at a distance of about 12 inches, the light is reflected into the eye. The pupil appears luminous, but the details of the fundus can not be seen. If there are opacities in the cornea, lens, or vitreous, they will appear as black spots in the brilliant pupil.

The localization of any opacity may be determined with some accuracy by the following method: If the observer's eye is moved so that the opacity which he sees lies nearly in line, let us say, with the lower edge of the pupil, when the patient's eye is turned upward the opacity, if it lies *back* of the pupil, will disappear behind the iris, or if it lies *in front*, it will appear to move upward from the edge of the pupil. The principle of parallax is here involved.

The behavior of the opacity with reference to a brilliant point of light which is the reflex from the anterior surface of the cornea, and which is referred to a point just back of the posterior pole of the lens, may be observed in the same way.

The vitreous should be carefully scanned for fixed or floating opacities while the eye is moved about. Lesions in the vitreous may be located and followed at different depths by bringing into place convex lenses of the ophthalmoscope.

III. The details of the fundus may now be investigated by what is known as the **direct method** or the **method of the erect image.** The patient and the light should be placed as just described, and the upper edge of the ophthalmoscope placed on the supraorbital ridge of the observer with the chin held down. The light is reflected into the pupil of the patient's eye and the observer approaches very near. If the accommodation is relaxed by an attempt to look at a distance with both eyes open, the fundus will come into view, if the refraction of both the patient and observer is normal. The erect image thus seen will be magnified about 15 times.

IV. **The Indirect Method or the Method of the Inverted Image.**—The light is thrown from the ophthalmoscope, held at a distance of about 18 inches, with a +3 D. lens before the aperture.

With the other hand the lens used for oblique illumination (+15 D.) is held about 2 inches from the patient's eye. An inverted image will form between the object lens and the ophthalmoscope. It is magnified about 5 times.

Choice of Method.—The method of the erect image is to be preferred for closer examination since the enlargement is greater but the field is small. The use of the ophthalmoscope for determining errors of refraction will be described under the head of Errors of Refraction.

The Fundus.—The ophthalmoscope opens to one's view a little more than the **posterior hemisphere** of the internal surface of the eyeball. It is often desirable to use a mydriatic to facilitate the examination. Homatropine hydrobromate (2 per cent.) or euphthalmin (5 per cent.) are the most suitable for this purpose. In carrying out a complete examination of the fundus the two landmarks which should be first found are the **optic nerve** (**optic disk, papilla**) and the **macula** (**yellow spot**). The former appears as an oval pinkish-white disk. A pit with sloping sides is in the centre (physiological excavation), with the mottled appearance of the lamina cribrosa at its bottom. A white ring of varying breadth (**scleral ring**) surrounds the disk, and outside of this more or less pigment (**choroidal ring**). From the nerve comes the **central artery of the retina** as a single trunk, or already divided, and it then divides and subdivides on the retina. The **veins** follow in general the same course as the arteries. The **color**, amount of **blood-supply**, sharpness of **outline**, and **swelling of the disk** should not escape notice. The presence of an excavation with sharp sides, over which the vessels seem to fall (**cupped disk**), may denote glaucoma. The other landmark—**the macula**—which is very difficult to see when the pupil is small, is an area of deeper color than the surrounding fundus, and shows a pit in its centre with a bright reflex (**fovea centralis**). An examination can not be thorough without a careful investigation of this region, whose integrity is so necessary to perfect vision. The **retinal vessels** should be followed from the nerve over the fundus and changes in them noted. The **general appearance** of the fundus is variable with the amount of pigment, depending on the complexion of the individual. When the **retinal pigment** is plentiful, a dark-red, mottled background is presented for the retinal vessels; when scanty, the choroidal vessels as well are seen as a network either lighter or darker than the background of choroidal pigment. Remembering that the retinal vessels are in the nerve-fibre layer, which is practically the inner layer of the retina, that the retinal pigment is the outer layer of the retina, and that the choroidal vessels lie in the **choroidal pigment**, the depth of any lesion may be made out from these landmarks. The fundus should be searched in all directions for lesions—such as *blood* in spots, splashes, or large areas, *white patches* of exudate, degeneration, or of exposed sclera. *Black masses* of pigment—retinal or choroidal,

Tension.—The proper way to test the hardness of the eyeball is by making the patient look down, and delicately, with the two first fingers, press through the upper lid until the eyeball is felt. By alternately pressing with either finger while the other finger is held fixedly in contact with the eyeball as the surgeon gets the "sense of fluctuation" a judgment of the tension is obtained.

Degrees of Tension.—An eye that is somewhat harder than the normal is described as having tension, T. +1. Decided rise of tension is recorded as T. +2; stony hardness as T. +3. Conversely, T. —1, T. —2, T. —3, denote varying degrees of softness. Instruments for recording the degree of tension have been devised, but are of little practical value.

Vision.—The sense of sight is divided into: I. Form-sense (acuity of vision); II. Color-sense; and III. Light-sense.

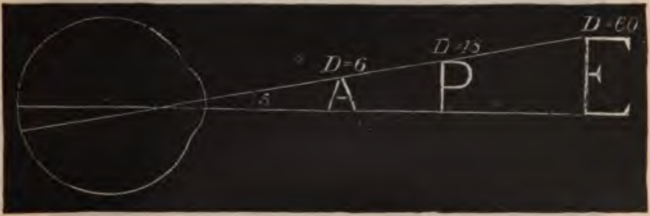
The form-sense may be classified as (*a*) *direct* or central vision, and (*b*) *indirect* or peripheral vision.

I. **Acuity of Vision.—Distance.**—In order to record with exactness the acuity of (*a*) *direct* or *central* vision the employment of letters has been found the most practical test. The universal method is to determine the smallest letters which the patient can read from a card containing letters of various sizes placed at a given distance. The construction of such a card is based upon the assumption that, with average acuity of vision, a patient should normally recognize a letter at any given distance when the height and breadth of that letter subtend an angle of 5′ of arc, the apex of which angle is at the patient's eye. In other words, if two lines are made to diverge from the eye forming an angle between them of 5′, letters fitted between these lines at different distances will vary in size, but will all form the same size image on the retina and be seen equally well. The letters on Snellen's card, which is usually employed, are all of a size determined upon the principle just stated and properly numbered for the distance at which they are placed in the angle.

Method of Recording Acuity of Vision.—Each eye should be tested separately. With the card placed in a good light a distance of 20 feet from the patient, the smallest letters which can be read are noted. The number over these, which denotes the distance in feet or metres at which they *should* be read, forms the denominator of a fraction whose numerator is the distance of the card from the patient—generally, as stated, 20 feet or 6 metres. Example: If the number of the line of smallest letters read is 40, the record will be $V. = \frac{20}{40}$. When vision is too poor to be tested with letters, the distance at which fingers may be counted should be noted. Or, vision may be reduced to perception of light (p. l.) or may be nothing $(V. = 0)$. For the illiterate, forms such as the spots of playing-cards or familiar objects may be employed instead of letters.

Near Point.—A card for the purpose of testing vision at the near point is constructed upon the same principles, but various sizes of printers' type are usually employed (Jaeger's). The record is made for the number of the type read and at the reading distance, 12 to 14 inches. The ability to change the refractive power of the dioptric media of the eye is called the **accommodation**, and lies in the action of the ciliary muscle, which in contraction relaxes the tension on the suspensory ligament of the lens, and the latter by its inherent elasticity becomes more convex, especially as to its anterior surface, and thus enables the focus for a near object to fall upon the retina. The test for the accommodation consists in observing the nearest point to the eye at which the print remains clear.

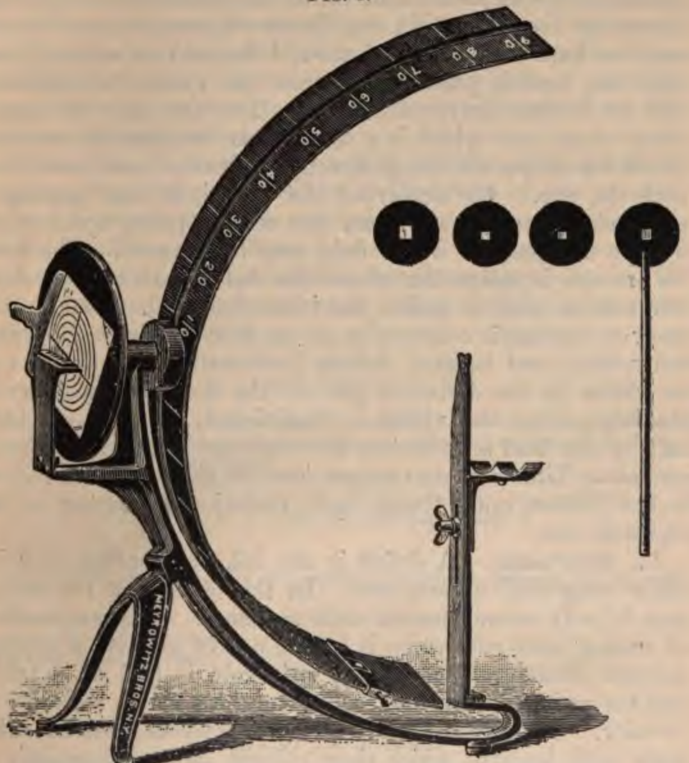
FIG. 3.



The visual angle.

Field of Vision.—The area of more or less distinct vision

FIG. 5.



Perimeter. The examination may be made with the carrier which moves along the semicircle, or the test-objects may be carried along this by means of dark disks attached to a long handle, each disk containing in its centre the test-object. The patient's chin is placed in the curved chin-rest; the notched end of the upright bar is brought into contact with the face, directly beneath the eye to be examined, which attentively fixes the centre of the semicircle. The other eye should be covered, preferably with a neatly adjusted bandage. The record-chart is inserted at the back of the instrument, and, by means of an ivory vernier, the examiner is enabled to mark exactly with a pencil the point on the chart corresponding to the position on the semicircle at which the patient sees the test-object. The various marks are then joined by a continuous line, and a map of the field is obtained.

and tracing with a piece of chalk the limits of the field while the eye is fixed upon a point directly in front. For exact measurements an instrument called the **perimeter** is employed. It consists essentially of a rest for the chin and a semicircular metal or hard rubber arc of about 1 foot radius, so fixed that when the head is placed in the rest the eye to be examined will be in the centre of the arc. The arc may be turned about on an axis which is a line drawn between the eye and the centre of the arc lying directly in front of and on a level with the eye. By revolving the arc about and passing a white object on it away from the central point, upon which the eye is fixed, the whole field may be covered. The field for one eye is called the monocular field of vision; the field common to both is called the binocular field. One should observe concentric contraction of the field of vision, irregular contraction, and isolated defects (scotomata). When there is no vision in the defective part of the field, the scotoma is absolute; when the vision is diminished, relative. If one-half of the field is defective, the condition is known as hemianopsia. This is due to causes local in the eye or to lesions in the chiasm, optic tracts, optic radiation, or cortex of the occipital lobe.

II. **Color-sense.**—A defect in the color-perception may be either congenital or acquired. In the latter case the defect may be only central (central color scotoma). The best method of testing color-perception is by the use of skeins of colored worsted (Holmgren's test). A red skein is laid on a table, and the patient is required to choose others which resemble it in color. For railroad and marine employeés tests are also made with lanterns which show colored lights (Thomson's or Williams' lanterns). Colors are not seen so far from the point of fixation as white—*i. e.*, the field for colors is smaller. Green has the smallest field; then red, blue, and white in the order named.

III. **Light-sense** is the power of the eye to appreciate variation in the intensity of illumination. An instrument called a **photometer** may be used in measuring differences of illumination. Diseases such as retinitis may affect the light-sense.

Muscles.—Only the external muscles of the eyeball are included under this caption. Although the methods of detecting error of motility will be referred to later, it is well to test the action of the muscles roughly at this point. The excursion of each eye, while following the finger in every direction, should be tested and limitations noted (paralysis or paresis). There should be no deviation of either line of vision from the object of fixation (strabismus).

If one eye is covered with a card and the other eye fixed upon any object, there should be no deviation of the covered eye, and it should make no movement when uncovered (cover-test for insufficiency). Both eyes should fix upon the same object and the two images should fuse (binocular vision). If both eyes do not fix upon the same object, double images are likely to result (diplopia).

QUESTIONS.

Describe the method of examining the conducting lachrymal apparatus.
State points to be observed in examining the lids, conjunctiva, and sclera.
What is oblique illumination?
Give the forms of conjunctival discharge and of congestion of the eyeball.
Describe examination of the cornea, anterior chamber, iris, and lens.
Explain the four methods of using the ophthalmoscope.
Describe the fundus.
Give test of tension.
Describe method of testing the acuity of vision at distance and at near point.
What is the field of vision?
How is color-sense tested?
Give superficial tests for the ocular muscles.

CHAPTER II.

DISEASES OF THE LACHRYMAL APPARATUS.

DISEASES of the lachrymal apparatus are **classified** as diseases of the secreting and diseases of the conducting portions.

DISEASES OF THE SECRETING PORTION.

The secreting portion consists of the *lachrymal gland*, including the *accessory gland of Rosenmüller*.

The **lachrymal gland** is rarely the subject of disease.

Acute nonsuppurative inflammation (dacryoädenitis), sometimes called "**mumps**," and **acute suppuration** (**abscess**), do occasionally occur. It may also be involved in **a chronic inflammation**, causing hypertrophy. A **cystic distention** of one of the ducts (*dacryops*) is also described. **Tumors** of the lachrymal gland are not unknown—*e. g.*, **carcinoma, sarcoma,** cysts, tuberculosis, and syphilis. It may become **prolapsed** downward, when it may be seen and felt. *Fistula* is of **rare** occurrence.

DISEASES OF THE CONDUCTING PORTION.

The conducting portion consists of the *puncta*, *canaliculi*, *lachrymal sac*, *lachrymal* (*nasal*) *duct*.

Malposition or **stenosis** (**atresia**) **of the puncta** or **canaliculi** may be congenital. The most common condition, however, is **displacement of the puncta** by turning out of the lids (**ectropion**), so that they do not lie against the eyeball. This is produced by thickening of the lid in chronic **diseases**, relaxation in old age and in facial paralysis, or by **cicatricial** contraction after traumatism. **Foreign bodies**, as an eyelash, leptothrix, or calcareous deposits and **wounds** may occlude the lumen.

Symptoms.—The natural result of stenosis or malposition is epiphora—the patient complaining of a watery eye, tears flowing on the cheek, especially in wind or in cold weather.

Treatment.—For congenital as well as acquired stenosis treatment is often unsatisfactory. If the punctum alone is affected, it may sometimes be opened, enlarged, or dilated with success. For malposition, due to hypertrophy of the lid, treatment consists in astringent applications to the conjunctival surface, such as nitrate of silver, 1 to 2 per cent. solutions.

DISEASES OF THE LACHRYMAL SAC.

The lachrymal sac has a small inlet through the **canaliculi** and an outlet through the nasal duct. Mechanical **interference** with the flow of tears from the sac into the **nose tends** to distention and disease of the sac. The diseases of the sac are **classified** as (*a*) *Chronic catarrhal dacryocystitis;* later (*b*) *Acute suppurative dacryocystitis* (*abscess of the lachrymal sac*) may supervene.

CHRONIC DACRYOCYSTITIS.

Synonym.—Blennorrhœa of the lachrymal sac.

Definition.—A chronic catarrhal inflammation of the mucous lining of the lachrymal sac.

Etiology.—The underlying cause is stricture of the nasal duct, usually accompanying nasal diseases, such as catarrh, polypi, or traumatic disturbance of the bone, with damming back of the tears and distention of the sac (mucocele). The stricture is generally either at the upper or the lower end of the duct. The mucous membrane of the sac thus becomes diseased from the presence of the accumulated discharge.

Subjective Symptoms.—The patient complains of epiphora and troubles referred to the lids, which come from the ever-present complicating chronic conjunctivitis.

Objective Symptoms.—The distended sac may be seen and felt as an elastic tumor. When pressure is made upon it, the contents usually pass out through the puncta, or are forced into the nose through the stricture of the nasal duct. The lids are red, swollen, showing more or less discharge at the edge from the conjunctivitis and blepharitis, and the caruncle is swollen.

Course.—Such a condition in the sac may exist for years, but abscess is always liable to occur, and the disease does not tend to improve. In long-standing cases necrosis of the adjoining bone may ensue.

Diagnosis.—The epiphora and discharge of mucopurulent fluid through the puncta on pressure over the sac make the diagnosis unmistakable.

Palliative treatment consists in the instillation of astringents—*e. g.*, zinc sulphate (0.3 per cent.) or alum (0.5 per cent.)—immediately after emptying the sac by pressure, and in treating the primary nasal disease especially in the region of the opening of the nasal duct. The sac may be syringed with astringent solutions, such as nitrate of silver (0.5 to 1 per cent.).

Operative Treatment.—I. Slitting the canaliculus and cutting the stricture in the nasal duct are accomplished as follows: A small, narrow, probe-pointed knife (canaliculus-knife), with a long, slightly curved shank, is introduced usually into the lower punctum. After entering with the point downward, the knife is turned in a horizontal direction and passed along the canaliculus, with the cutting edge inward toward the eyeball, while the lid is drawn in the opposite direction with the finger and held quite taut. The knife should be passed through the lachrymal sac until it meets the lachrymal bone. With the point pressed against the bone, the handle should be raised until vertical, and the canaliculus thus fully slit, while the point is pushed down through the nasal duct into the inferior meatus of the nose. The direction of the knife should be toward the groove between the ala of the nose and the cheek. The injection into the sac of a solution of adrenalin chloride (1 : 10,000) and cocaine hydrochlorate (2 per cent.) will facilitate the operation by their vasoconstricting action. Immediately after withdrawing the knife the whole tract must be syringed with a mild antiseptic solution, such as boric acid (3 per cent.). The next step consists in passing a probe (usually the largest possible) through the nasal duct into the nose, and allowing it to remain in place for a few minutes. Bowman, Theobald, and Weber probes are the common styles, and they vary in diameter from 1 to 4 mm. To prevent the stricture from reforming, the probing should be continued at first every other day, and later, at longer intervals, for several weeks or months, depending on the character and behavior of the stricture. The operation and

Fig. 6.

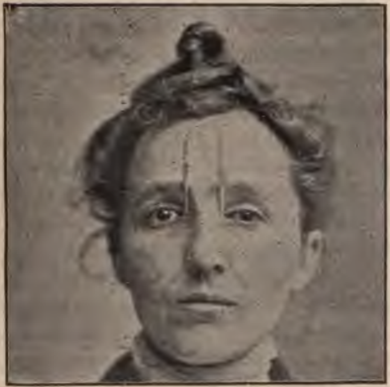
Weber's canaliculus-knife.

probing are painful, and the results are not always satisfactory, at least as far as the epiphora is concerned, although the slitting of the canaliculus alone will usually prevent danger of abscess. In infants operative measures should be undertaken cautiously, since many cases recover under palliative treatment.

II. The introduction into the nasal duct of lead or silver styles which are left in place indefinitely was formerly much practised.

III. In obstinate cases and in those where operative procedures on the eyeball are contemplated and infection feared, the sac should be destroyed. It is exposed by a skin incision and cauterized or dissected out. The epiphora is not distressing, but excision of the lachrymal gland has been advocated.

FIG. 7.



Bowman's probes in position. (Reeve.)

ABSCESS OF THE LACHRYMAL SAC.

Synonyms.—Purulent dacryocystitis; Phlegmon.

Definition.—Acute suppurative inflammation of the sac and surrounding tissues.

Etiology.—Chronic dacryocystitis is always the forerunner of abscess. Infection is the immediate cause.

Pathology.—The pyogenic germs which are always present in the sac may excite acute inflammation in the sac and walls, with the same pathogenesis as elsewhere in the body.

Subjective Symptoms.—The patient who has been troubled with epiphora or dacryocystitis is suddenly attacked with severe throbbing pain in the region of the sac.

Objective Symptoms.—The skin becomes red, tense, swollen, and tender. The lids also are swollen, and there may be constitutional disturbances. If not incised, the abscess points and bursts on the skin surface and may leave a permanent fistulous tract connecting with the sac.

Diagnosis.—The involvement of the lachrymal sac is established by the situation of the abscess and the history of epiphora, but the condition might be mistaken for abscess of the lid or for processes involving the bone in the vicinity.

Prophylactic treatment consists in treating the dacryocystitis.

Active Treatment.—When the abscess is forming, hot fomentations or poultices are indicated. When the tumor softens, it should be promptly and freely incised through the skin and the cavity syringed and packed with gauze. The wound is then dressed daily on general surgical principles. If excessive granulations form, they may be treated by scraping or by nitrate of silver stick. When all reaction has subsided, the canaliculus should be slit and the nasal duct probed if possible, in order to prevent recurrence, which is liable to take place.

QUESTIONS.

What lesions may occur in the secreting portion of the lachrymal apparatus?

What are the causes, symptoms, and treatment of malposition or stenosis of the puncta and canaliculi?

How are diseases of the lachrymal sac divided?

What are the causes of chronic dacryocystitis?

What are the symptoms and complications?

Describe the palliative and operative treatment.

Describe abscess of the lachrymal sac.

What results may follow stricture of the nasal duct?

CHAPTER III.

DISEASES OF THE LIDS.

BLEPHARITIS MARGINALIS.

Synonyms.—Blepharitis ciliaris; Tinea tarsi; Blepharo-adenitis.

Definition.—A chronic inflammation of the edge of the eyelids accompanied by congestion, thickening, ulceration, and formation of crusts and scales.

Etiology.—The underlying cause is often a conjunctivitis with improper care in cleansing the roots of the lashes where the discharge collects. Errors of refraction are indirectly responsible in some cases. Other causes are improper mode of life, depression in the general health, late hours, smoke, and dust. It complicates lachrymal disease and may follow the exanthemata.

Dermatological Classification.—The disease assumes the form of (*a*) *seborrhœa*, where the sebaceous glands are affected, forming scales or yellow crusts; (*b*) *sycosis*, in which case there is suppuration in the hair-bulbs; (*c*) *eczema*, being often associated with eczema of the face in children.

Clinical Classification.—The disease is commonly divided into *ulcerative* and *nonulcerative* blepharitis.

Symptoms.—The congestion along the roots of the lashes is often the only symptom (hyperæmia marginalis). This comes and goes at the least provocation, such as eyestrain, late hours, exposure to wind, etc. In more marked form there will be formation of scales and crusts (squamous blepharitis). In other cases minute pustules (pustular blepharitis) are found, and when these rupture, hard crusts and scabs form, under which ulcerations are found (ulcerative blepharitis).

Course.—The disease, which is most common in children, extends over many years and ultimately results in complete loss of the eyelashes (madarosis), with the edge of the lid thickened and everted (tylosis).

Treatment, which is tedious, consists in correcting errors of refraction, reforming the mode of life, and attending to constitutional disturbances.

Local Treatment.—Strict cleanliness should be observed. The edges of the lids must be washed with soap and water or solutions of borax or bicarbonate of sodium until free from crusts. Ointments may then be applied, such as the following:

℞ Hydrargyri oxidi flavi,	0.15;
Petrolati,	10.00.

M. et ft. ung.
Sig.—Rub on the edge of the eyelids every night.

One may also use ointments of hydrargyrum ammoniatum (1 per cent.), hydrargyrum rubrum (1 per cent.), or ichthyol (5 per cent.). In ulcerative cases nitrate of silver solutions (1 to 2 per cent.) or even the stick of nitrate of silver are indicated. When pustules form about the hair-follicles, the cilia should be pulled out. Astringents such as zinc sulphate (0.3 per cent.) or nitrate of silver (1 per cent.) should be used to cure the complicating conjunctivitis.

HORDEOLUM.

Synonym.—Stye.

Definition.—An acute circumscribed suppurative inflammation beginning in the glands at the margin of the lid.

Etiology.—The presence of blepharitis, disturbances of digestion, impaired vitality, and eyestrain. The immediate cause is infection with pyogenic germs.

Pathology.—On account of the numerous glandular structures, the edge of the lid is a favorable place for the entrance of infection. It is also liable to slight abrasions by rubbing.

Symptoms.—Itching and burning sensations followed by a red and swollen area at the edge of the lid. Later the abscess forms, points, and discharges. There is often extensive œdema of the lid out of proportion to the size of the lesion.

Course.—Styes run their course in from three days to a week or more, and are often repeated one after another. At times one will not reach the suppurative stage, but become aborted or remain as a hard swelling ("blind stye").

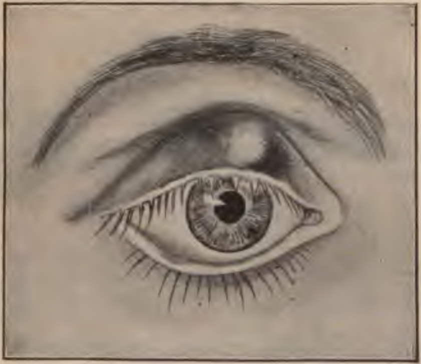
Treatment.—The digestive tract should be treated and constitutional treatment instituted. Bits of ice held against the beginning stye may abort it. Ointments of red or yellow oxide of mercury (1 per cent.) may be applied. Later hot fomentations will encourage suppuration. Early incision will cut short the course.

CHALAZION.

Synonyms.—Tarsal tumor; Meibomian cyst.

Definition.—A chronic affection of the Meibomian glands with the formation of a hard swelling in the lid.

FIG. 8.



Chalazion. (Reeve.)

Etiology.—Chalazia are regarded by some as due to infection, but in many cases the direct cause is the stoppage of a Meibomian duct and the accumulation of discharge in the gland.

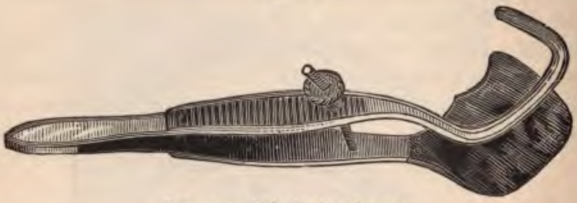
Pathology.—The process seems to be a chronic inflammation with the production of granulation-like material originating in the Meibomian gland. Under the microscope these tumors in the typical stage show scant connective-tissue stroma with many round cells and without true capsule.

Subjective Symptoms.—The patients usually have little complaint except on account of the appearance of the swelling. The inflammatory symptoms which may occur at the beginning and the roughened condition of the lid occurring later may create more or less irritation.

Objective Symptoms.—A chalazion appears as a round or elongated tumor varying in size up to that of a walnut. It is firmly adherent to the tarsal cartilage with the skin usually freely movable over it. On the conjunctival aspect a dark spot generally shows in the tarsus.

Course.—A chalazion may begin with symptoms of hordeolum and later lose inflammatory signs, or may appear without inflammation. At times the adjoining tissue is involved in chronic inflammatory infiltration. Later the centre of the chalazion breaks down into a clear or yellowish fluid, and the degeneration continues until the whole mass becomes a cyst with thickened walls. Generally the fluid escapes through a spontaneous opening on the inner aspect of the lid. This soon closes, and about it granulations form sometimes as large polypoid masses. Chalazia are likely to be multiple and their course is protracted. They often spontaneously disappear.

FIG. 9.



Desmarres' chalazion forceps.

Treatment.—Hot fomentations may be tried with massage.

Radical treatment consists in curetting or in excision. In the former procedure, a vertical incision is made on the inner or conjunctival side, and the granulation-masses are thoroughly scraped out with a small sharp- or serrated-edged spoon (Meibomian scoop). The sac refills with blood, which is absorbed in a few days. This operation may require repetition. A more satisfactory method consists in making a liberal incision under aseptic conditions through the skin over the tumor parallel to the edge of the lid. The mass may then be dissected out, using the knife and scissors. The chalazion with the region of the lid about it should be clamped off by an instrument (Desmarres' clamp) consisting of a horn plate and a ring on opposite arms like a forceps. This will prevent troublesome hemorrhage and fix the tumor. One or more stitches are generally desirable after the bleeding has stopped. An antiseptic powder, as iodoform or aristol, may be used as a dressing and covered with court plaster.

ENTROPION.

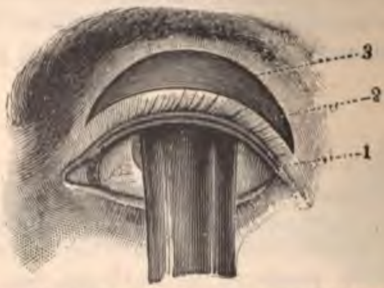
Definition.—A condition in which the edge of the eyelid is turned inward against the eyeball.

Etiology.—The common cause of entropion is cicatricial contraction of the palpebral conjunctiva (*cicatricial entropion*). This is generally produced by trachoma, but may be due to wounds or burns. Entropion may also be due to a spasmodic condition of the orbicularis of the lower lid in elderly people with lax condition of the soft parts (*spasmodic entropion*). A similar condition appears in children when there is great photophobia such as accompanies ulcers of the cornea.

Symptoms.—The most serious consequence of entropion is the brushing of the lashes against the eyeball (trichiasis), which leads to irritation and congestion of the eyeball and ulceration of the cornea.

Treatment.—For **spastic entropion**, if temporary, the skin of the lid can be pulled down toward the cheek by adhesive plaster or may be folded by taking a long stitch. In **trichiasis** with the presence of only a few inturned lashes, these may be pulled out at intervals or destroyed by electrolysis. But in marked cases of cicatricial entropion **operative treatment** is indicated. The simplest method consists in excising an elliptical piece of skin of the lid and closing the opening with sutures (**Graefe's operation**). This procedure is applicable to mild cases, and may also be applied to persistent spastic entropion. For marked cases many different operations have been employed. They are for the most part variations of the following procedures: An elliptical piece of skin is excised from the lid with the underlying muscle. If there is much incurving of the tarsal cartilage, a wedge-shaped groove is cut horizontally in the middle of the tarsus and sutures are passed through the skin and cut edges of the tarsus (**Streatfeild-Snellen's operation**). Instead of grooving the tarsal cartilage, a good effect is obtained by splitting the margin of the lid lengthwise, being careful to leave all the cilia and hair-bulbs on the outer flap. This may be left to granulate, but the skin wound is sutured (**Jaesche-Arlt's operation**). A narrow strip of skin (Hotz) or mucous membrane (Van Millengen) without a pedicle may be fitted into the groove at the lid margin and will attach itself. In connection with the above operations it is of great advantage at times to lengthen the palpebral fissure at the outer canthus (**canthoplasty**). This is done by a single cut with the scissors and by passing sutures between the cut edges of the conjunctiva and skin.

FIG. 10.



Jaesche-Arlt's operation for trichiasis: 1, intermarginal incision; 2, 3, positions of the second and third incisions, between which the integument is removed. (Czermak.)

ECTROPION.

Definition.—A rolling out of the lid—the opposite of entropion.

Etiology.—This condition is largely due to thickening of the conjunctiva and margin of the lid, or to cicatricial contraction of the skin following wounds, burns, ulceration, or caries of the orbital margin. It also occurs from relaxation of the lid in elderly persons or in paralysis of the orbicularis.

Symptoms.—Ectropion is unsightly, exposing as it does more or less of the conjunctiva. Epiphora, chronic conjunctivitis, and irritation are complained of. The lower lid is the more frequently affected.

Treatment.—When recent, moderate in degree, and due to hypertrophy of the conjunctiva, applications of nitrate of silver (1 to 4 per cent.) to the conjunctival surface may give relief. In advanced and marked cases the hypertrophied tissue may be excised. When ectropion is due to cicatricial contraction, relief is obtained only from some form of operation. **Snellen's operation** consists in passing a suture with two needles, each of which is entered inside the lower lid and comes out through the skin on the cheek, leaving a loop on the conjunctival surface. The ends are then tightly tied on the cheek, and the cicatricial bands, which result, tend to roll the lid inward. In marked cases a **plastic operation**, generally by the use of sliding flaps, is necessary in order to relieve the tension of the cicatrix on the lid. Shortening the palpebral fissure by joining together a portion of the upper and lower lids at the outer canthus (**tarsorrhaphy**) may be advisable.

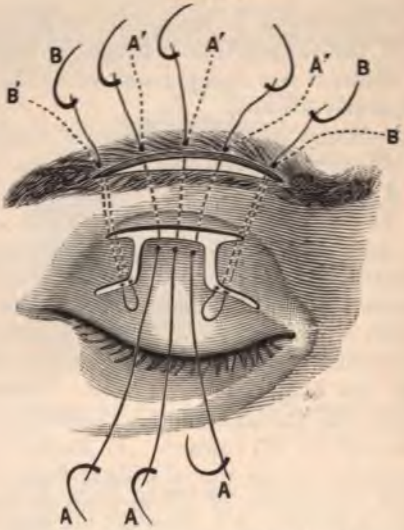
PTOSIS.

Definition.—A drooping of the upper lid.

Etiology.—Ptosis may be due to paralysis or insufficiency of the levator palpebræ. It occurs either in the **congenital form**, when it is usually bilateral, or in the **acquired form**, when it is generally unilateral. In the latter case especially, it may be associated with paralyses of other muscles supplied by the third nerve, such as are due to syphilis or diseases of the brain. There are still other cases due to mechanical causes, such as increased volume of the lid with trachoma, tumors, etc. Forceps injuries at birth are not unknown as a cause.

Symptoms.—The upper lid will cover the pupil in marked ptosis, and the patient will attempt to raise the lid by contraction of the frontalis muscle, holding the head well back, thus giving the characteristic expression.

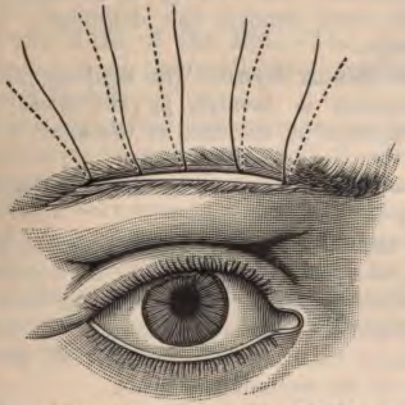
FIG. 11.



Panas' operation: A, A′, central sutures; B, B′, lateral sutures. (Nettleship.)

Treatment.—If ptosis is due to paralysis or to mechanical causes, the **constitutional** and **local measures** may avail. If the ptosis is permanent, and especially in the congenital cases, **operative procedures** are indicated. The underlying principle involved in nearly all the operations is the attempt to connect e upper lid more directly with the occipitofrontalis muscle, and thus exaggerate the action of that muscle in raising the lid. The simplest method (**Graefe's operation**) is to excise an elliptical piece of skin with the underlying tissue of the lid and suture the cut edges of the wound. The **Pagenstecher operation** consists in using a suture with two needles, each of which is passed from the edge of the lid up under the skin and brought out near the eyebrow. The ends are then tied, and the cicatricial bands, which form along the lines of the sutures, serve to connect the lid with the frontalis muscle. These two methods are applicable only to the mild cases, and are not always highly satisfactory. The **Panas method** consists in dissecting a flap of skin from the upper lid in the form of a tongue which points upward. This is drawn up with sutures underneath the undermined skin of the eyebrow.

FIG. 12.



Panas' operation, after. (Nettleship.)

INJURIES OF THE LIDS.

Wounds.—Especial **danger** attends wounds of the lids from the fact that disturbance in their function of covering the eyeball is serious. All wounds **should be** carefully s both the conjunctival and skin side. They heal kindly, as a rule.

Burns.—When extensive, burns are likely to cause contractions and adhesions, resulting in entropion, ectropion, or symblepharon.

MISCELLANEOUS DISEASES OF THE LIDS.

Emphysema.—An inflation of the subcutaneous tissue of the lids with air takes place when air is forced through a fracture of the walls of the orbit, which establishes a communication with the nose or accessory sinuses. The appearances are those of simple œdema, but a peculiar crackling sensation is experienced on palpation. The history of an injury and violent blowing of the nose confirm the diagnosis. It disappears under a bandage.

Ecchymosis (Black Eye).—The settling of blood in the lid after contusion is favored by the loose subcutaneous tissue. In fractures at the base of the skull it occurs in the lower lid.

Treatment.—Ice applications in the first few hours should be followed by hot fomentations.

Œdema of the lids is a symptom common after injuries, insects' bites, inflammatory action, like hordeolum or dacryocystitis, and occurs as angioneurotic œdema, from urticaria, in myxœdema, nephritis, cardiac disease, or is idiopathic. It may be associated with severe conjunctivitis and deep-seated inflammations of the eyeball and orbit.

Abscess of the Lid.—Generally traumatic. Should be incised when pus has formed.

Syphilis only rarely appears on the lids. It may occur as chancre, mucous patch of the conjunctiva, gumma, or tertiary ulcer.

Lupus is found on the lid, and **vaccine ulcers** are described.

Blepharospasm is an involuntary contraction of a part or whole of the orbicularis muscle.

Varieties.—It occurs as clonic or tonic spasm. In its simplest form it appears as a fibrillary twitching of a part of the lid which sometimes annoys and alarms the patient. It may take the form of continual winking (nictitatio). This usually occurs in children as the result of irritation from the conjunctiva and is often choreic in nature. Spasmodic contractions of the orbicularis are also associated with tic douloureux, or may be of hysteric origin.

Blepharospasm may also appear in diseases producing irritation, such as phlyctenular keratitis.

Lagophthalmos is a condition in which the lids can not be completely closed, and is due to exophthalmos, injuries, paralysis of the orbicularis, or contractions of the lid.

Phthiriasis Ciliorum.—*Pediculi pubis* occasionally deposit eggs on the lashes and the insects themselves will be found buried in the lid margin.

Diseases of the Skin of the Lid.—The skin is the seat of various diseases, such as erythema, eczema, erysipelas, herpes zoster, and syphilis.

Epicanthus is a congenital deformity in the form of a fold of skin which extends from the inner end of each eyebrow to the side of the nose, covering the inner canthus and giving the appearance of a very broad bridge of the nose. It may be accompanied by ptosis. Treatment consists in removing a piece of skin from the bridge of the nose.

Coloboma of the lid is a congenital defect of rare occurrence. It is a cleft in the lid similar to cleft-palate and harelip. It may also be traumatic origin.

TUMORS OF THE LID.

Varieties.—Beside chalazion and hordeolum a number of **benign tumors** are found on the lid, including verruca (wart), molluscum contagiosum, xanthelasma, dermoid cysts, milia, vascular tumors, cutaneous horns, and small transparent cysts at the edge of the lids (glands of Moll).

Molluscum contagiosum appears as a rounded elevation formed by the hypertrophy of a sebaceous gland and duct.

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CHAPTER IV.

DISEASES OF THE CONJUNCTIVA.

CONJUNCTIVITIS (OPHTHALMIA).

Classification.—

Conjunctivitis	Catarrhal	Acute, Chronic.
	Purulent	Ophthalmia neonatōrum, Gonorrhœal conjunctivitis.
	Granular	Trachoma.
	Phlyctenular.	
	Membranous	Croupous, Diphtheritic.

ACUTE CATARRHAL CONJUNCTIVITIS.

Synonyms.—Acute mucopurulent conjunctivitis; Acute contagious conjunctivitis; "Pink-eye."

Definition.—An acute catarrhal inflammation, especially of the palpebral conjunctiva, characterized by congestion, swelling, and mucopurulent discharge.

Etiology.—Exposure to wind, dust, and smoke, or presence of irritating foreign substances. Koch-Weeks bacillus, pneumococcus, streptococcus, staphylococcus, and Morax-Axenfeld diplobacillus are responsible for infection in different cases. It may be associated with the exanthemata. Occurs in epidemic form, especially in spring and fall.

Varieties.—A number of varieties exist, but clinically they may be classed under two heads: *simple* and *infectious*, the latter being generally due to the Koch-Weeks bacillus and called *acute epidemic conjunctivitis.*

Subjective Symptoms.—Patients complain of stiffness of the lids, photophobia, epiphora, burning, and sensations of a foreign body. There is no actual pain, but considerable discomfort, especially in the evening.

Objective Symptoms.—The lids are swollen and red. The conjunctiva of the globe is more or less deeply congested, and that of the lid is thickened, congested, and rough. Occasionally there are subconjunctival hemorrhages. The mucopurulent discharge is collected at the roots of the lashes or lies on the surface of the conjunctiva. The lids are stuck together in the morning. The vision is only slightly affected by the mucous discharge on the surface of the cornea, which is otherwise clear, although minute ulcerations of the cornea are occasionally seen.

Diagnosis is easily made by the presence of mucopurulent discharge, deep congestion involving the conjunctiva (especially palpebral), clear vision, and absence of pain. **Differential diagnosis** will be found under the head of acute glaucoma.

Course.—The disease usually attacks one eye a few days in advance of the other. The first stage of congestion lasts a few hours or a day, and is followed by the stage of discharge, which continues a few days to a week or more. Most cases recover quickly, but the greatest danger is in chronic conjunctivitis and blepharitis remaining as sequelæ.

Prophylactic Treatment.—As epidemic conjunctivitis is contagious, and often attacks a whole family, the promiscuous use of towels, etc., should be avoided.

Active Treatment.—In the first stage applications of gauze or cotton taken directly from ice are indicated, together with a wash, such as boric acid solution (3 per cent.), chlorine water (50 per cent.), or formaldehyde (1 : 5000). In addition to this, when the discharge appears, astringents are called for, such as applications of nitrate of silver (1 per cent.). An efficient prescription is as follows:

℞	Zinci sulphatis,	0.10;
	Alum,	0.15;
	Aquæ destill.,	25.00.

M. Sig.—One drop into eye twice or three times a day.

The lids should be anointed with vaselin at night to prevent sticking with discharge. The common practice of applying poultices of tea-leaves or bread and milk is to be condemned, and the eyes should not be bandaged.

CHRONIC CATARRHAL CONJUNCTIVITIS.

Definition.—A chronic catarrhal inflammation usually confined to the palpebral conjunctiva.

Etiology.—May follow an acute conjunctivitis. It is caused by dusty atmosphere, night work, and late hours; may be due to eye-strain from errors of refraction or improper use. It may be associated with nasal catarrh or with constitutional disturbances, such as rheumatism and gout.

Subjective Symptoms.—There is complaint of burning, smarting, itching, sensations of a foreign body, blurring, lachrymation, photophobia, dryness, heaviness, sleepiness, and discharge, which sticks the lids together in the morning.

Objective Symptoms.—The palpebral conjunctiva varies in appearance from slight injection to deep congestion, and the surface may be studded with granulations or cheesy deposits (lithiasis), and be more or less thickened. The edges of the lids are hyperæmic, and there is watery or mucopurulent discharge, with excoriations of the skin, especially at the outer and inner canthus. Occasionally a white flocculent discharge will be found, which is chemically a soap, with sodium as a base.

Course.—The disease, which is extremely common, runs a protracted course, being subject to exacerbations at intervals. Both eyes are usually affected.

Systemic Treatment.—Unfavorable conditions should be corrected.

Local measures consist in the use of astringents. In mild cases boric acid in saturated solution, zinc sulphate (0.3 per cent.), alum (0.5 per cent.), tannic acid (1 per cent.), chlorate of potassium (0.3 per cent.), tinc. opii (50 per cent.), or protargol (5 per cent.), may be tried. In marked or obstinate cases applications of silver nitrate (1 to 2 per cent.), every day or every other day, are indicated. Daily application of alum crystal is an excellent remedy. Nasal catarrh should be treated. The disease is often intractable, and frequent changes in treatment are desirable.

CHRONIC FOLLICULAR CONJUNCTIVITIS.

Synonym.—Follicularis.

Definition.—A chronic disease of the palpebral conjunctiva, characterized by the presence of hypertrophied follicles, with few or no inflammatory signs.

Etiology.—Common in youth, especially in strumous children who live under unfavorable hygienic surroundings. It may be infectious.

Pathology.—The "follicles" are masses of lymphoid tissue resembling true trachoma granules.

Subjective symptoms are those of mild, chronic catarrhal conjunctivitis.

Objective Symptoms.—The granulations appear as small, round, pale elevations, confined to the fornix and nasal portion of the *lower lid*, and to the conjunctiva at the edge and over the extremities of the tarsal cartilage of the upper lid.

Diagnosis.—Follicular conjunctivitis is difficult at times to differentiate from trachoma, and is regarded by some as belonging to the same category. (See Trachoma.)

Treatment is the same as for simple chronic conjunctivitis.

OPHTHALMIA NEONATORUM.

Synonyms.—Purulent conjunctivitis in the infant; Acute blennorrhœa; Gonorrhœal conjunctivitis in the new-born.

Definition.—A severe conjunctivitis in the new-born, usually due to the gonococcus of Neisser and characterized by *purulent* discharge.

Etiology.—While mild cases of conjunctivitis in the new-born may be occasioned by less virulent forms of infection from the parturient canal or from outside causes, and are generally classed under this head, the majority of the severer cases are due to gonococcus infection.

Symptoms.—The first symptoms are swelling and redness, usually of both eyes, occurring the second or third day after birth. Very soon the discharge begins to appear, and shortly becomes creamy pus, which runs from the eye when the swollen lids are parted. As the disease advances the conjunctiva of the lids is thickened, red, and velvety, and that of the eyeball is œdematous.

Complications.—If the pus is allowed to remain in the conjunctival sac, the cornea may become hazy and ulcers appear. If an ulcer perforates, the iris is likely to be caught in the opening and in the resulting scar (**adherent leucoma**). The cornea, weakened by inflammation, may later bulge and produce **anterior staphyloma**. Or, the whole eye may become involved in an inflammation which results in its destruction.

Course.—The disease lasts from two to six weeks, often leaving chronic conjunctivitis.

Prognosis.—If seen before corneal ulcerations set in, the vast majority of these cases recover.

Treatment.—Ophthalmia neonatorum is a prolific cause of blindness, and its nature, prevention, and treatment should be thoroughly understood.

Prophylaxis.—In public institutions and at times in private practice the *Crédé* method should be employed. It consists in dropping a 2 per cent. solution of nitrate of silver into the conjunctival sac of the infant at birth. Salt solution should be used immediately afterward. The frequency with which an active catarrhal conjunctivitis follows the use of 2 per cent. solutions of nitrate of silver has led to the employment of 1 per cent. strength in the hands of many observers. The prophylactic value is equally great and the undesirable secondary catarrh of the conjunctiva is much less common than with the original Crédé strength. Such practice has greatly reduced the percentage of ophthalmia neonatorum in lying-in hospitals. It is well also to douche the vagina and cleanse the eyes of the new-born with a mild antiseptic solution, such as boric acid (3 per cent.).

Active Treatment.—When the disease is established, energetic treatment should be instituted. The lids should be gently separated and the discharge flushed out with distilled thickened granular and congested conjunctiva (chronic blennorrhœa).

Prognosis is decidedly grave. The majority of the well-established cases result in loss of or in serious damage to the eye involved.

Prophylactic Treatment.—Individuals with gonorrhœa should exercise the greatest care to prevent infection of the eyes. The healthy eye must be covered by a watch-glass framed with surgeon's plaster and closely applied about the eye (Buller's shield).

Active treatment of the disease is entirely similar to that of a severe case of ophthalmia neonatorum.

Metastatic Gonorrhœal Conjunctivitis.—Cases of gonorrhœal urethritis complicated by systemic infection manifesting itself as arthritis may suffer from conjunctivitis of metastatic origin. There is little or no discharge, but small corneal ulcers sometimes appear.

TRACHOMA.

Synonyms.—Granular conjunctivitis; Granulated lids; Egyptian ophthalmia.

Definition.—A moderately contagious disease in which the palpebral conjunctiva is occupied by new tissue, usually in the form of small elevations, which after a prolonged existence pass over into cicatrices.

Etiology.—The disease belongs to the lower classes, among whom sanitary conditions are poor and cleanliness is not strictly observed. It is not unlikely that the origin of the disease is a microörganism, but its existence has not been established. It is more common among certain races—*e. g.*, Jews and Irish.

Pathology.—The trachoma granulations are composed of lymph-corpuscles with scanty connective-tissue stroma and incomplete capsule. They are imbedded in the conjunctiva.

Trachoma is commonly **classified** as follows:

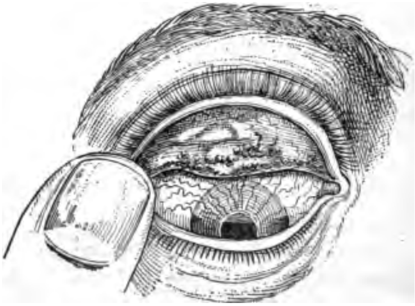
I. **Papillary trachoma,** in which the conjunctiva is hypertrophied and folded, forming granulations (papillæ) especially over the tarsal cartilages. The appearance is velvety or coarsely granular. This form occurs also after blennorrhœa and from other causes. It is not, properly speaking, true trachoma.

II. **Granular, follicular,** or **true trachoma,** appears as round, transparent bodies (granules, trachoma follicles), especially in the fornices. Each granule is a mass of lymph-corpuscles, without true capsule.

III. **Mixed form,** where the two are associated. This is the most common.

Symptoms.—The disease is divided into three stages: I. Onset. II. Full development. III. Cicatrization.

FIG. 13.



Typical granular lid and beginning cicatrization, with pannus. (Berry.)

I. The *onset* may be *acute, subacute,* or *chronic.*

a. **Acute Onset (Acute Trachoma).**—An uncommon form, in which there are rapid swelling, redness, and irritation of the lids. The conjunctiva is much thickened, red, and granular (papillæ), and there is considerable mucopurulent or purulent discharge. This condition resembles severe acute conjunctivitis, or even gonorrhœal conjunctivitis.

b. **Subacute Onset.**—The conjunctiva is moderately swollen, deeply congested, and roughened. As the swelling disappears the true granules are seen.

c. **Chronic Onset (Noninflammatory Trachoma).**—There is little or no discomfort. The lids may appear slightly swollen. On everting the lower lid masses of granulations roll out from the fornix, and the upper lid shows a tarsus covered with granulations, and the retrotarsal fold will be found filled with trachomatous masses. Congestion is usually absent.

II. **Stage of Full Development.**—The disease usually assumes the mixed form with papillæ and granules. There is considerable irritation, lachrymation, photophobia, and slight mucopurulent discharge. The tarsal cartilages are covered with coarse and fine granulations, and the fornices are filled with trachoma masses. The conjunctiva is moderately congested and thickened. This condition may exist for years with exacerbations, but gradually passes over into the cicatricial stage.

III. **Stage of Cicatrization.**—White or bluish areas or lines (cicatrices) begin to appear among the granular masses, and by degrees the conjunctiva presents a shining surface streaked with white cicatricial bands. The fornices are practically obliterated in extreme cases, and the conjunctiva becomes dry (xerosis). During the periods of full development and transition complications make their appearance.

Palpebral Complications.—The inevitable contraction which follows when granulation-tissue is converted into cicatricial tissue is accompanied by disastrous results in the eye, for the shrinking of the inner surface of the lids bends the tarsal cartilage and rolls the margin of the lid inward (entropion), and the lashes sweep over the cornea (trichiasis).

Corneal Complications.—A superficial vascular keratitis (pannus) appears with ulcerations and infiltration, especially on the upper half of the surface of the cornea, with bloodvessels running down over the cornea from the conjunctiva above. Pannus may cover the whole cornea and leave dense opacities seriously affecting the vision. Ulcers may be deep and perforate.

Deeper complications may appear in the form of iritis, cyclitis, and even panophthalmitis, from which phthisis bulbi results.

Course. Trachoma is essentially a chronic disease, often beginning in youth and continuing for years.

Differential Diagnosis.—*Chronic follicular conjunctivitis* and *trachoma* sometimes resemble each other. The former occurs in youth. The granulations are small, round, in rows, occurring especially on the nasal half of the conjunctiva of the lower lid and edge and extremities of the cartilage of the upper lid. The disease is amenable to treatment and the granulations disappear without leaving a trace. Trachoma, on the contrary, is a progressive disease, lasting for many years. The granulations are, as a rule, larger, less prominent, more uniformly distributed, and at last disappear, leaving cicatrices.

Prophylactic Treatment.—Children in schools and asylums should be protected against this common contagious disease. Each child's towel, and handkerchiefs should be provided, and cleanliness enforced. Cases should be, to a certain extent, isolated.

Medicinal Treatment.—The sovereign remedy is sulphate of copper. The crystals should be rubbed over the diseased portion of the lid every day or every other day, not forgetting to go well under the tarsal cartilage into the retrotarsal fold of the upper lid. The eyes should be immediately bathed with cold water. Nitrate of silver (1 per cent. solution) is a good substitute for short periods. If applied too long, it will cause permanent staining of the conjunctiva (argyrosis, argyria). Tannin and glycerin (60 grains to the ounce) is of slight value. Also, mercuric chloride (1 : 1000) may be used. Applications of yellow oxide of mercury ointment (1 per cent. for home treatment) are useful in later stages. Atropine may be necessary in corneal and iridic complications.

Surgical treatment consists in mechanically crushing the granulations. The roller-forceps of Knapp is generally used for this purpose. The granulations are caught between two grooved rollers and their contents squeezed out, as in a wringer. The operation should be thoroughly performed under a general anæsthetic. The lids should be treated with applications of copper sulphate for some time after the expression. The operation is indicated in noninflammatory trachoma and in many cases with well-marked granulations. Other forceps sometimes employed are those of Noyes and Prince.

Grattage is a method in which the granulations are broken by rubbing with a stiff tooth-brush. It should be used with care.

PHLYCTENULAR CONJUNCTIVITIS.

Synonyms.—Scrofulous ophthalmia; Eczematous conjunctivitis.

Definition.—A disease characterized by the development of small papules or pustules on the bulbar conjunctiva.

Etiology.—The disease occurs in strumous children and in those who are ill-fed. It is allied to eczema in its nature and often is associated with it on the face. It is rarely seen in the adult. It is commonly accompanied by nasal catarrh. The same lesions occur on the cornea (phlyctenular keratitis). It is probably due to a microörganism.

Subjective Symptoms.—The child complains very little if the phlyctenules are not near the cornea. There is some photophobia, irritation, and lachrymation.

Objective Symptoms.—On the bulbar conjunctiva will be found one or more elevations in the form of papules or pustules surrounded by a circumscribed area of congestion. Later the pustules may break down and form ulcers. A favorite site is at the margin of the cornea. It is then clinically known as *phlyctenula marginalis.* Phlyctenules often form in rapid succession, each lasting a week or more. Relapses are common.

General Treatment.—Since this disease is a manifestation of constitutional derangement, tonics such as iron, quinine, and cod-liver oil are indicated. Proper food, exercise, and fresh air are prescribed.

Local Treatment.—Once or twice a day a small quantity of yellow oxide of mercury ointment (1 per cent. in vaselin) should be placed inside the lids and thoroughly rubbed about with the lids closed. Atropine (0.5 per cent. solution) should be ordered if the cornea is affected. Calomel may be dusted into the eye. The nose should receive appropriate treatment.

MEMBRANOUS CONJUNCTIVITIS.

Membranous conjunctivitis is a rare disease.

Two forms are described under the heads of *croupous* and *diphtheritic*.

Croupous conjunctivitis is the more common variety, and may occur as a complication in severe forms of conjunctivitis in children, complicating infectious diseases, or as the result of superficial burns. It presents a membranous deposit which if removed leaves a bleeding surface.

Treatment is same as for gonorrhœal conjunctivitis.

Diphtheritic conjunctivitis is a rare affection due to the Klebs Loeffler bacillus. The reaction may be comparatively slight but usually the lids are greatly swollen, reddened, tense, and stiff. A dirty yellow diphtheritic membrane is found on the conjunctiva when it is possible to evert the lids. Constitutional symptoms of diphtheria are present. The necrosis following the infiltration of the conjunctiva results in a granulating surface which cicatrizes and deforms the lid. The cornea and whole eye are likely to suffer.

Treatment.—Cases should be isolated and the other eye protected. Diphtheritic antitoxin should be employed. Locally the treatment is the same as for gonorrhœal conjunctivitis.

INJURIES OF THE CONJUNCTIVA.

Foreign bodies frequently lodge on the palpebral conjunctiva. **Wounds** and **burns** with lime, acids, or metals occur. The contraction which follows extensive destruction of the conjunctiva, or the uniting of adjacent surfaces of the conjunctiva of the eyeball and lid, produces adhesions which are known as **symblepharon**. It may be partial at the fornix or in the form of bands, leaving the fornix free, or it may completely unite the lid to the eyeball.

Treatment.—If symblepharon is simply cut and the lid freed from the globe, it is impossible to prevent recurrence. Sliding flaps of healthy conjunctiva should be made to cover the cut surfaces and prevent reattachment. Flaps of skin or mucous membrane without a pedicle are rarely successful. The most favorable cases are the bands which allow a probe to be passed around through the fornix.

MISCELLANEOUS DISEASES OF THE CONJUNCTIVA.

Spring Catarrh.—**Synonyms.**—Vernal catarrh; Spring catarrh; Saemisch spring catarrh; Conjunctivitis æstiva.

Definition.—A peculiar, uncommon disease characterized by the presence of nodular masses about the periphery of the cornea and extending a little on to it, associated with hard, flat, pale granulations on the conjunctiva of the upper lid.

Etiology is unknown.

Symptoms.—The disease appears either with circumcorneal lesion or granulations on the lid alone or both together. It usually occasions very little discomfort except some irritation, photophobia, and sensation of a foreign body. It occurs in the spring, summer, and autumn, but nearly or entirely disappears in the winter. As the corneal lesions are all confined to the periphery they do not affect the vision, and after recovery usually no traces are left.

Treatment is unsatisfactory except in relieving irritation.

Xerosis is a term applied to two different affections. The name is used to designate: I. The condition of dryness seen in advanced cicatricial contraction of the conjunctiva such as occurs after the ravages of trachoma (atrophy of the conjunctiva, parenchymatous xerosis).

Treatment of this condition is of no avail.

The term also applies to: II. A disease which shows white plaques on the surface of the bulbar conjunctiva (epithelial xerosis), due probably to the presence of xerosis bacillus. This membranous, greasy appearing deposit may be scraped off. In infants (infantile xerosis) it is associated with marasmus and usually complicated by indolent ulcers of the cornea. These latter cases always end fatally.

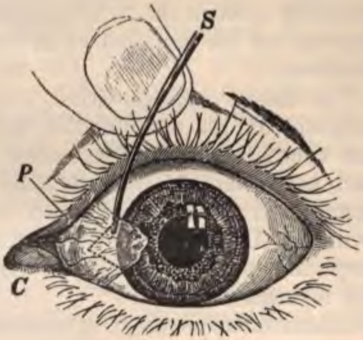
Treatment in adults consists in scraping off the membrane and applying antiseptics, such as mercuric chloride (1 : 1000).

Pterygium. — **Definition.** — A superficial membranous growth having its base near the inner or outer canthus and extending with its point toward the centre of the cornea.

Etiology.—This growth is formed of hypertrophied conjunctival tissue and thought in some cases to be an extension of the pinguecula (page 63).

Symptoms.—The patient complains very little except when the pterygium is well advanced toward the centre of the cornea and astigmatism is produced or the vision cut off.

FIG. 14.



Pterygium. (Fuchs.)

The wing-like membrane occurs most often on the nasal side, may be on the temporal, but is very rare above or below. When progressive, it is thick and somewhat congested. When nonprogressive, it is dry, thin, and nonvascular. Pseudopterygium is somewhat similar in appearance, but produced by burns or ulcers.

Course.—It extends over many years and may spontaneously cease advancing at any time.

Treatment.—Surgical treatment is necessary. If the pterygium is dissected off the cornea, which may readily be done, and then cut off, it will recur. It is necessary to cover the loss of tissue on the conjunctival surface with a conjunctival flap which will extend to the edge of the cornea. An effective method (transplantation) consists in dissecting off the pterygium from the cornea and sclera, leaving the base attached and burying its apex beneath the undermined conjunctiva below.

Pinguecula is a small yellow elevation in the ocular conjunctiva usually on the nasal side and near to the cornea. It is a hyaline degeneration of the conjunctiva and is of common occurrence in middle and late life. Occasionally it becomes inflamed. No treatment is required.

Tuberculosis of the conjunctiva is uncommon. **Primary lesions** have been recorded. **Secondary affections** in the form of nodules or ulcerations occur and are occasionally associated with nasal disease.

Parinaud's disease somewhat resembles tuberculosis of the conjunctiva. It is accompanied by enlargement of the preauricular and cervical glands with constitutional symptoms. It is a rare disease.

Syphilis of the Conjunctiva.—Chancre and **Gumma** are of rare occurrence.

Amyloid conjunctivitis is characterized by waxy masses in the fornix.

Ecchymosis of the conjunctiva is extravasation of blood beneath the conjunctiva due to rupture of bloodvessels. Caused by traumatism, paroxysms of coughing, as in whooping-cough, and spontaneously in elderly persons whose bloodvessels are sclerosed. No treatment is necessary.

Lymphangiectasis of the Conjunctiva.—Clusters of transparent blisters in the bulbar conjunctiva due to dilatation of lymph-spaces.

Chemosis of the Conjunctiva.—Œdema of the bulbar conjunctiva occurring in violent inflammatory conditions of the eye.

Pemphigus of the Conjunctiva.—Bullæ form, and are followed by cicatricial tissue which destroys the whole conjunctiva. "Essential shrinking" of the conjunctiva is an allied process. The conditions are extremely rare.

Tumors of the Conjunctiva.—Cyst, angioma, dermoid, lipoma, fibroma, papilloma, epithelioma, and sarcoma have been described.

QUESTIONS.

Give the classification of conjunctivitis.

Give the differential diagnosis between acute conjunctivitis, iritis, and glaucoma.

Describe the treatment of acute catarrhal conjunctivitis.

Describe chronic catarrhal and chronic follicular conjunctivitis.

Give definition, etiology, symptoms, complications, and treatment of ophthalmia neonatorum.

Describe gonorrhœal conjunctivitis.

What are the varieties of trachoma? Give the pathology, the stages, the complications, and differential diagnosis between trachoma and follicular conjunctivitis?

What two forms of membranous conjunctivitis occur?

Define spring catarrh, xerosis, pterygium, symblepharon, and pinguecula.

Mention other rarer diseases of the conjunctiva.

What microörganisms cause diseases of the conjunctiva?

What is the prognosis for the various diseases of the conjunctiva?

CHAPTER V.

DISEASES OF THE CORNEA.

ULCER OF THE CORNEA.

Definition.—Superficial loss of substance accompanied by more or less infiltration of adjacent cornea.

Etiology.—Depression in the general health is an underlying cause, or there may be poor nutrition of the cornea itself. Ulcers are more common among the poorer classes. They often begin in an abrasion produced by a foreign body. May be associated with diseases of the conjunctiva or lachrymal apparatus.

Pathology.—The process is a necrosis of the superficial layers of the cornea due to infection. Among the microorganisms which have been found are staphylococcus, streptococcus, pneumococcus, Morax-Axenfeld bacillus, gonococcus, and aspergillus.

Varieties.—A *broad distinction* may be made between **simple** or **nonprogressive ulcers** and **infected** or **progressive ulcers.** Simple ulcers are small, may follow abrasions with clean foreign bodies, are amenable to treatment and not associated with deep-seated complications. They are due to some mild form of infection. Infected ulcers, on the contrary, may follow injuries with dirty foreign bodies, often resist all treatment, tend to spread, and are accompanied by complications. They are due to some active form of infection—*e. g.*, pneumococcus.

The **important clinical varieties** are:

Phlyctenular ulcer (see Phlyctenular Keratitis).

Traumatic ulcer. Following abrasions or wounds.

Serpent ulcer (Saemisch ulcer). An infected ulcer with advancing crescent-shaped edge.

Dendritic ulcer. An infected ulcer branching in shape, and superficial. May be malarial in origin.

Ring ulcer. Attacking the periphery of the cornea and sometimes completely encircling it.

Indolent ulcer (absorption ulcer). Shows little or no inflammatory signs. Occurs in the aged, in marasmic infants, and in debilitated subjects.

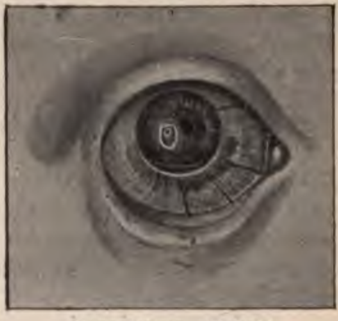
Catarrhal ulcer. Accompanying catarrhal conjunctivitis.

Subjective Symptoms.—The patient complains of photophobia, lachrymation, sensations of foreign body, more or less pain especially in infected ulcers. There is defect in vision when the ulcer is over the pupil.

Objective Symptoms.—The eyeball shows a ring of pink congestion about the cornea (circumcorneal), together with more or less congestion of the conjunctiva. Ulcers vary greatly in appearance, but in general with oblique illumination show a superficial loss of substance in the cornea with a gray opacity (leucocyte infiltration) in the adjacent tissue. The form of the ulcer may be irregular, circular, crescentic, dendritic (branching), or punctate.

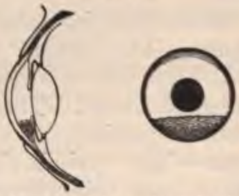
Course.—Simple ulcers usually heal kindly in a week or two by throwing off the necrotic tissue and replacing it with connective tissue. Infected ulcers may progress by spreading superficially, by undermining healthy tissue with an advancing edge or in branching lines. Or, they may sink deeply into the substance of the cornea and even perforate. Sometimes the progress is by colonies, which break out in all directions. When the healing process has begun, bloodvessels will often be found running inward from the periphery of the cornea.

FIG. 15.



Corneal ulcer. (Sichel.)

FIG. 16.



Hypopyon, seen from the front, and in section, to show that the pus is behind the cornea. (Nettleship.)

Complications.—Opacities of the cornea are the inevitable result of filling of the ulcer with connective tissue. The effect on the vision depends on their density and situation.

If a deep ulcer perforates into the anterior chamber, there is danger of the iris being caught in the resulting scar, in which case the condition is called adherent leucoma. The cornea weakened by ulceration may later bulge, forming staphyloma, which may or may not contain the prolapsed iris.

In infected ulcer a quantity of pus is sometimes seen in the lower part of the anterior chamber (hypopyon). This is composed of cells thrown off from the iris and endothelial layer of the cornea as the result of irritation. At times, also, pus will accumulate on the substance of the cornea (formerly called onyx), which may break down into an abscess. Iritis is a common complication of infected ulcers. Iridocyclitis and even panophthalmitis may follow, with destruction and shrinking of the eye (phthisis bulbi).

Diagnosis.—The presence and extent of the loss of substance may be visibly demonstrated by the use of a drop of 2 per cent. solution of fluorescin, which stains the exposed area a bright green. An ulcer may be known from an old opacity by the presence of circumcorneal congestion, subjective symptoms, loss of substance, or roughened surface of the cornea.

Preventive Treatment.—After abrasions of the cornea a mild antiseptic should be given in the form of eye-drops—boric acid, saturated solution, or mercuric chloride (1 : 5000). In removing a foreign body aseptic instruments should be employed. Complicating conjunctivitis or dacryocystitis should be treated.

Constitutional Treatment.—It is important to treat the general condition if infected ulcers are associated with debility, syphilis, or improper mode of life.

Local Treatment.—Atropine (1 to 2 per cent.) should be instilled three to six times a day. Eserine is sometimes of use, but tends to produce iritis. Hot fomentations, repeated according to the severity of the case, and an antiseptic should be prescribed, such as boric acid (3 per cent.), permanganate of potassium (1 : 3000), or chlorine water (50 per cent). Indolent, phlyctenular, and healing ulcers should be stimulated by rubbing in the eye yellow oxide of mercury ointment (1 per cent. in vaselin). Calomel or iodoform may also be dusted into the eye. If there is much irritation, holocaine (1 per cent.) may be instilled. A pressure-bandage is indicated unless there is much conjunctival discharge. In infected ulcers more energetic treatment is required to arrest the infection. In addition to the above the base and edges should be scraped with the eye under cocaine (5 per cent.), and nitrate of silver (1 per cent.) or tincture of iodine (full strength) should be applied by means of cotton on an applicator once a day or once every other day. The actual cautery, such as a red-hot strabismus-hook or electrocautery, may be tried. The application should be in the form of numerous punctures at the edge of the ulcer. If hypopyon is present, it is sometimes wise to split the ulcer into the anterior chamber with a knife (**Saemisch operation**). Dense opacities left after ulcerations are sometimes conspicuous, and may be tattooed by the introduction of India ink with needle-pricks. An artificial pupil may sometimes be made through the iris if the pupil is covered by the opacity.

INTERSTITIAL KERATITIS.

Synonym.—Diffuse parenchymatous keratitis.

Definition.—A chronic inflammation involving the parenchyma of the cornea, characterized by deep-seated opacities and circumcorneal congestion.

Etiology.—The common form of this disease appears in children and is due to congenital syphilis. It rarely occurs in acquired syphilis. It has been known to occur in utero. The disease may also be due to malaria, rheumatism, gout, tuberculosis, and rickets, or may be idiopathic.

Pathology.—The cornea normally has no bloodvessels, and inflammatory action consists in infiltration of leucocytes, although occasionally in this disease a deep-seated vascularization may take place at the periphery.

Subjective Symptoms.—The patients complain of moderate pain, photophobia, and poor vision.

Constitutional Objective Symptoms.—Congenital syphilis is usually easily recognized in other parts of the body by characteristic signs, such as the so-called Hutchinson teeth. The incisors of the permanent set are small, furrowed from side to side, peg-shaped, narrowed, and notched at the extremity. The face, especially at the angles of the mouth and forehead, is scarred and wrinkled from early, even intra-uterine, ulcerations. The head is large and square. The lymph-glands are enlarged. The bridge of the nose is flat, and there are chronic aural and nasal troubles.

Local Objective Symptoms.—The opacities of the cornea may begin at any point, but usually at the periphery as a thin, gray cloud. The opacities spread and often completely cover the cornea, becoming at times a dense white or yellowish and mottled. Deep-seated bloodvessels will enter from the sclera and produce a dense red spot called "salmon patch." The surface of the cornea is sometimes dull and rough and "steamy." There is little or no tendency to ulceration, although it is possible for the cornea to weaken and bulge, forming staphyloma.

Course.—Both eyes are usually affected, although it may be at different times. The disease occurs between the ages of five and fifteen, though sometimes seen as late as thirty years. It is slow in its course, lasting from two months to a year or more. The opacities clear often to a remarkable degree, but in severe cases the vision is more or less impaired. Relapses are common. Complications in the form of inflammations of the uveal tract (iritis, cyclitis, and choroiditis) often occur in severe cases.

Internal Treatment.—Mercury and iodide of potassium, in doses suited to the age of the patient, are indicated, although the cases of congenital syphilis may do as well with tonics, such as iron and cod-liver oil, with out-of-door life.

Local Treatment.—Atropine (1 per cent.) should be instilled three times a day during the active period, and hot fomentations applied regularly for at least fifteen minutes four times a day. Stimulation by rubbing in the yellow oxide of mercury ointment is of value, if there is not too much irritation. Dark glasses should be worn. Injections of normal salt solution under the ocular conjunctiva (subconjunctival injections) may be tried. In acquired syphilis energetic antisyphilitic treatment is necessary.

PHLYCTENULAR KERATITIS.

Definition.—The disease is of the same nature and due to the same causes as phlyctenular conjunctivitis.

Varieties.—*a.* Fascicular keratitis. An ulceration which presents a curved, advancing edge of infiltration with a bunch of bloodvessels passing to it from the corneal edge. This often continues across the cornea and leaves a row of permanent opacities. *b.* Multiple ulcers, with more or less superficial vascularization.

Symptoms.—The young patient suffers greatly from photophobia and buries the head in the pillow. There is tonic blepharospasm, and the lids are separated with difficulty.

Treatment.—Atropine (1 per cent.) t. i. d. Yellow oxide of mercury ointment (1 per cent.) rubbed in at night. If there is great blepharospasm, the face may be plunged into a basin of cold water. Constitutional treatment is the same as for phlyctenular conjunctivitis.

STAPHYLOMA.

Synonym.—Ectasia.

Definition.—A bulging of the cornea or sclera, not due to swelling or thickening of the tissues. Staphyloma occurs in corneæ which have been the subject of disease which has weakened their resisting power to internal pressure, such as ulcerations, abscesses, or injuries. It is called **partial** or **total staphyloma** depending upon the extent of the cornea involved. When total and very prominent, the lids can not close. The eye is usually blind from previous inflammation, and sometimes the eyeball itself is shrunken.

Treatment.—For total staphyloma, operation consists in abscission of the protrusion and suturing the edges together; this is done for cosmetic reasons. The operation is usually safe, although serious inflammation has been known to follow.

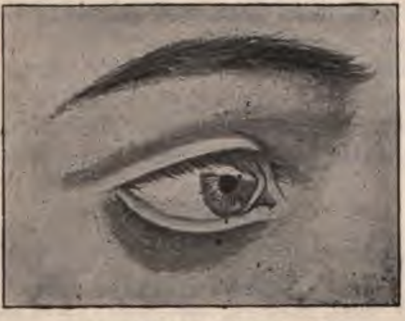
KERATOCONUS.

Synonym.—Conical cornea.

Definition.—A peculiar disease consisting in gradual bulging of the transparent cornea into a conical form with the apex at or near the centre. The process may begin at any period of life, but usually in youth, progresses slowly for many years without signs of inflammation, and may become stationary at any time.

Symptoms.—The patients complain of increasing defect in vision. On examination, in marked cases the conical form of the cornea is very evident, especially when viewed from the side. In slight cases a reflex from the window is greatly distorted by lengthening on every side from the apex of the protrusion. The change in the curve of the cornea creates a high degree of astigmatism and myopia. A gray opacity may appear at the apex of the cone and ulcerate.

FIG. 17.



Conical cornea. (Dalrymple.)

Treatment.—The progressive character of the condition must be established by extended observation. If *nonprogressive*, strong cylinders often greatly improve the vision and should be carefully selected. If *progressive*, treatment should begin at once, and consists in destroying a portion of the cornea at the apex of the cone in the hope that the contraction following will flatten the protrusion. The best method is to burn deeply with the electric cautery, using a small electrode with a flattened end.

INJURIES OF THE CORNEA.

Abrasions.—The anterior epithelial layers of the cornea are easily torn off by a foreign body. If the object is clean, the cells will reform in a few hours under a bandage. If the abrasion is infected by the foreign body or by bacteria already in the conjunctival sac, an ulcer will result, which may lead to disastrous consequences. The pain, photophobia, and lachrymation are quite intense after an abrasion, owing to exposure of the nerves.

Treatment.—An antiseptic, such as mercuric chloride (1 : 5000) or boric acid (saturated solution), should be prescribed and the eye bandaged.

Burns of the cornea may be by hot water, steam, metals, acids, and alkalies. If superficial, being aseptic, they heal quickly; if deep, the scars may affect the vision.

Treatment.—Atropine (1 per cent.) and a bandage are indicated.

FOREIGN BODIES.

The cornea, on account of its soft structure and its exposed position, is a favorite lodging-place for foreign bodies, such as particles of dust, cinders, coal, emery, and steel. For the detection of a foreign body it is necessary to use the oblique illumination and sometimes a magnifying-glass in addition.

Treatment.—The eye should be thoroughly anæsthetized by dropping in a solution of cocaine (4 per cent.) or holocaine (1 per cent.) twice with ten minutes' interval. The eyelids should be spread apart with the fingers or with a speculum, and the foreign substance picked out of the cornea by the use of a sterilized blunt spud or foreign-body needle. Great care must be exercised not to injure the cornea more than is absolutely necessary. Some foreign bodies, especially emery and bits of steel, may be quite deeply buried, and must be tacked carefully yet boldly. If the cornea should be perforated and the foreign body lie partly in the anterior chamber, it must not be pushed further in. An eye drop of boric acid (saturated solution) should be prescribed and the eye bandaged.

PERFORATING WOUNDS OF THE CORNEA.

Wounds which open the **anterior chamber** and allow the aqueous to escape are likely to be complicated by falling in of the iris (prolapse of the iris). If the wound is not infected and not too extensive or contused, it will heal in a few days, and when the iris is not prolapsed only a scar will result. This may or may not affect the vision, according to its situation. If there is prolapse of iris, the healing will not be so rapid, and danger of subsequent inflammation will be incurred. A fistulous opening may remain, which may also happen in perforating ulcers.

Treatment.—The *wound* should be cleansed with an antiseptic solution (bichloride of mercury 1 : 5000), which should be continued, together with atropine (1 per cent.) and light pressure-bandage. Ice applications may be employed during the first day or two to control reaction.

Treatment of the prolapsed iris is important. If the wound is clean and the case is seen within forty-eight hours of the injury, the iris should be pulled through the wound with iris-forceps and cut off close to the cornea with scissors. The cut edges of the iris must be carefully replaced within the anterior chamber by a spatula. If the wound is infected, the iris should not be cut, since this procedure exposes the tissues to infection, which otherwise they might escape. If the case is over forty-eight hours old, adhesions to the wound have formed, which render separation of the iris difficult, and to leave the cut edges of the iris in the wound opens a path for infection to the interior of the eye.

MISCELLANEOUS DISEASES OF THE CORNEA.

Superficial Keratitis.—**Synonym.**—Vascular keratitis.

Definition.—A term used to signify a superficial inflammation such as that which complicates trachoma, otherwise known as pannus. It is characterized by the presence of bloodvessels and infiltration on the surface of the cornea.

Treatment consists in attacking the trachoma, but in severe cases the bloodvessels may be cut off by scraping the periphery of the cornea, or the galvanocautery may be used for the purpose.

Vesicular Keratitis.—A number of diseased conditions of the cornea presenting vesicle formations are classed under this head.

Herpes corneæ is a peculiar recurrent eruption of small vesicles on the surface of the cornea lasting for a few hours and accompanied by sensations of a foreign body, pain and irritation, which pass away when the vesicle ruptures. It sometimes follows an abrasion or injuries of the cornea.

Keratitis Bullosa.—Characterized by large bullæ, occurring usually in a diseased eye.

Herpes Zoster Ophthalmicus.—When herpes affects the fifth nerve, it may attack the cornea as well as the skin of the face. The corneal eruption usually leaves scars.

Treatment of vesicular keratitis is in general similar to that of ulcer of the cornea.

Filamentous Keratitis.—Shows threads attached at one end to an ulceration.

Keratitis profunda is a deep-seated central interstitial keratitis occurring in the adult and sometimes confounded with keratitis of specific origin. The cause is generally unknown, but it may be due to exposure, rheumatism, or malaria.

Local treatment is same as for interstitial keratitis.

Sclerosing keratitis accompanies scleritis as a dense white permanent opacity resembling the normal sclera.

Treatment is the same as for scleritis.

Ribbon-shaped Keratitis.—**Synonym.**—Transverse calcareous film.

Definition.—A grayish-white band extending horizontally across the cornea. It is hard to the touch of an instrument, since it contains lime. It occurs in eyes which have been diseased or are degenerated.

Treatment consists in scraping off the film.

Neuroparalytic keratitis is due to lesion of the trigeminus which cuts off the nerve-supply of the cornea. Characterized by anæsthesia, ulcerations, and necrosis caused by trophic changes and undetected foreign bodies.

Posterior punctate Keratitis.—**Synonym.**—Descemitis.

Definition.— A condition in which minute deposits occur on the lower part of the posterior surface of the cornea in the form of a triangle with the base down. It is a manifestation of disease of the uveal tract—*i. e.*, iris, ciliary body, or choroid (see Serous Iritis).

Superficial punctate keratitis is characterized by numerous small elevated opacities of the anterior layers of the cornea accompanied by congestion and irritation of the eye.

Tumors of the cornea are of rare occurrence. They are found mostly at the limbus, and dermoid fibroma, papilloma, epithelioma and sarcoma have been described.

Pigmentation of the Cornea.—The cornea may be stained with blood-pigment (hæmatin, hæmatoidin), or from the presence of iris or steel in the eye (**siderosis**).

Arcus Senilis (**Gerontoxon**).—A zone of opacity at the periphery of the cornea, but with a narrow zone of clear cornea between it and the sclera. It is a fatty degeneration, and generally found in elderly persons.

QUESTIONS.

Mention the varieties of corneal ulcers.
Give course, complications, and treatment.
Describe interstitial keratitis.
Describe phlyctenular keratitis.
Define other forms of keratitis.
Define staphyloma and keratoconus.
State rule for treatment of prolapse of iris in perforating wounds of the cornea.
What is arcus senilis?
What antiseptics are used in treating the cornea?

CHAPTER VI.

DISEASES OF THE SCLERA.

SCLERITIS.

Definition.—A localized inflammation characterized by the presence of more or less elevated, congested, and discolored patches in the sclera.

Etiology.—The disease occurs in adults who are the subjects of constitutional disorders, such as syphilis, rheumatism, malaria, gout, or tuberculosis. It may be idiopathic.

Varieties.—**Episcleritis**: Involves the superficial layers of the sclera. **Scleritis proper**: Where the whole depth is affected. A clinical variety of episcleritis is a mild recurrent form called episcleritis fugax. The elevated patches of specific origin may be called gummatous scleritis.

Symptoms.—I. **Episcleritis.** There is usually only a moderate amount of pain, photophobia, and irritation. A bright-red or violet, slightly elevated patch appears on the sclera not far from the cornea. The congestion is seen to be formed, not alone by the conjunctival vessels, but by those of the sclera underlying. There are frequent relapses. II. **Scleritis.** This is a more serious form. There may be considerable pain, tenderness, photophobia, lachrymation, and general irritation. There are elevated, red, yellow, or violet areas in the sclera, which, recurring, may extend about the whole cornea. The deeper structures—iris, ciliary body, and adjacent cornea—are often involved.

Complications.—Scleritis, iritis, keratitis, and cyclitis—a process affecting the anterior segment of the eyeball—is known as **anterior uveitis.** When the sclera and cornea alone are affected, it is called **sclerokeratitis.** The sclera may be thinned by inflammation and bulge from internal pressure, producing staphyloma. Glaucoma may ensue Patches of scleritis leave permanent dark-bluish spots at the site of the lesion, which should not be confounded with congenital pigmentation.

Constitutional treatment should be given according to the established cause. In idiopathic cases sodium salicylate, gr. x three or four times a day (although some advocate very large doses), or potass. iodide, gr. x t. i. d., is indicated.

Local Treatment.—Hot fomentations. Atropine (1 per cent.), t. i. d. Subconjunctival injections of salt solution or bichloride of mercury (1 : 5000) may be tried.

STAPHYLOMA.

Definition.—A bulging of the sclera not due to thickening. It **occurs** after scleritis; also in diseases of the choroid and ciliary body accompanied by high tension, or in weakening of the cornea or sclera after injury. It appears as dark bluish elevations (sometimes called ectasiæ).

Posterior staphyloma is a bulging of the sclera about the optic nerve, and is associated with high myopia. (See page 132.)

Treatment.—For anterior and equatorial staphyloma with increased tension iridectomy may be indicated; but if the eyes are destroyed by inflammation and are unsightly or painful, enucleation is necessary.

INJURIES OF THE SCLERA.

The sclera, with the cornea, is exposed to injury from **foreign bodies.** Injuries with blunt objects may produce **rupture of the eyeball,** which, beside the lacerated wound usually near the cornea, is often accompanied by internal injuries, such as rupture of the iris, dislocation of the lens, detachment of the retina, rupture of the choroid, or intraocular hemorrhage. The eyeball is soft and the vision is seriously affected. Rupture of the sclera, without break of the conjunctiva, is possible.

Incised wounds, when large, are accompanied by more or less loss of vitreous and prolapse of the ciliary body and choroid. The greatest danger beside that arising directly from the injury is from infection, which may produce an iridocyclitis or panophthalmitis, both of which may end in shrinking of the eyeball (phthisis bulbi). When the wound is in a zone occupying about one-fourth of an inch outside the periphery of the cornea (known as the "ciliary region" on account of the fact that the ciliary body lies beneath it), the injury has a further significance. Such a wound, followed by a chronic iridocyclitis, gives the conditions which may produce sympathetic disease in the other eye—sympathetic ophthalmia. If a foreign body is retained within the eye, the case is further complicated. (See page 118.)

Treatment.—*Small* wounds should be treated with pressure-bandage, an antiseptic (bichloride 1 : 5000), and atropine (1 per cent.). In *extensive* wounds, say over 10 mm., sutures should be passed through the scleral edges of the wound and others through the conjunctiva. These wounds sometimes heal remarkably well. If the ciliary body or choroid is prolapsed, it should be cut off. When an injury has evidently completely destroyed the eye, it should be enucleated at once.

QUESTIONS.

What are the varieties of scleritis?
Describe each.
Define staphyloma of the sclera.
Give differential diagnosis between episcleritis and phlyctenular conjunctivitis.
Describe injuries of the sclera and give treatment.

CHAPTER VII.

DISEASES OF THE IRIS.

IRITIS.

Varieties.—In point of *duration* and *severity* iritis may be divided into acute, subacute, and chronic. As regards *etiology*, into syphilitic, rheumatic, gonorrhœal, traumatic, tuberculous, secondary, sympathetic, and idiopathic. On a *thologic basis*, into plastic, serous, and purulent.

ACUTE PLASTIC IRITIS.

Definition.—An inflammation of the iris, characterized by congestion, small pupil, and posterior synechiæ.

Etiology.—The disease occurs in the secondary stage of syphilis—*i. e.*, from the second to the eighteenth month—and is rarely seen in the tertiary stage. Rheumatism is the next most frequent cause. It may occur with gonorrhœal rheumatism, but not usually at the same time. It may appear in diabetes, gout, or from traumatism, or may be idiopathic. It may also be secondary to inflammation of adjacent tissues—*e. g.*, in keratitis or scleritis.

Pathology.—The iris is congested, swollen, and infiltrated with round cells. There is an exudate of round cells, fibrin, and pigment-cells, which fills the anterior chamber and glues the edge of the pupil and back of the iris to the anterior capsule of the lens.

Subjective Symptoms.—The patient complains of more or less severe pain in the eye, forehead, and temple, especially in the early hours of the morning. There are fear of light and lachrymation. The vision is affected, and there may be some constitutional symptoms.

Objective Symptoms.—On examination the lids are found to be swollen and red. The eyeball shows the typical circumcorneal or ciliary congestion, with some congestion of the conjunctiva. The cornea under oblique illumination is seen to be hazy, and under careful examination the posterior surface will often be found studded with minute dots. The anterior chamber is cloudy, and there may be some deposits in the lower part. Rarely blood may be seen in the anterior chamber. The anterior surface of the iris has lost its fine and delicate details. It looks muddy, and has changed to a dirty color as compared with the other eye. The pupil is small, and scarcely moves to stimulus of light. It is more or less filled with cloudy exudate. If a drop of a mydriatic, such as atropine (1 per cent.), be instilled, the pupil will show irregular dilatation on account of the fact that at different points the pupillary edge of the iris is held to the lens by exudate (**posterior synechiæ**). If these be torn off by the action of the mydriatic, it is common to find a ring of pigment corresponding to the position of the edge of the iris before treatment was begun. On account of the hazy condition of the media the fundus is usually obscured. A form of the disease known as **spongy iritis** occurs especially after injuries and operations which open the anterior chamber. It is characterized by a gelatinous sharply defined mass of exudate in the anterior chamber, which resembles a partly opaque, dislocated lens. It often disappears with great rapidity.

Course.—The disease may occur at any time of life, but is uncommon in children, except as a secondary condition. It begins acutely and lasts from one to six weeks.

Prognosis.—When seen early, the prognosis is excellent if proper treatment is carried out. There is a tendency to recurrence.

Complications.—If the disease is severe, there may be formed numerous synechiæ and more or less deposit in the pupillary area. If the blocking of the pupil is complete, it is known as **occlusion**; if the synechiæ completely bind down the iris yet leave the pupil clear, the condition is called **exclusion.** In the latter circumstances circulation from behind the iris through the pupil is impeded, and the iris bulges forward into the anterior chamber, except at the pupillary edge; hence the name **crater-shaped pupil** (iris bombé). Glaucoma is likely to follow, and the condition must be relieved by iridectomy. After repeated attacks of iritis the iris may become atrophic and immovable. Cataract may also form. Adjacent tissues may be involved in the inflammatory process, and the conditions are designated by the following terms, which are self-explanatory: iridocyclitis, iridochoroiditis, keratoïritis, anterior uveitis. (See page 76.) In syphilitic iritis yellow nodules form at the pupillary margin or at the periphery. Gummata may develop in tertiary syphilis.

Diagnosis.—The pain, especially at night, the ciliary congestion, muddy iris, small pupil, and especially posterior synechiæ establish the diagnosis.

Differential Diagnosis.—See Acute Glaucoma.

Treatment.—Patients should be kept in a darkened room, and, if the attack is severe, in bed.

Constitutional treatment should be instituted according to the cause. Syphilis must be treated energetically with mercury protiodide ($\frac{1}{4}$ gr.), bichloride ($\frac{1}{25}$ gr.), inunctions of blue ointment, or hypodermatic injections of bichloride. Potassium iodide may also be given (gr. x, increased). In rheumatic cases it is well to give the patient a saline cathartic at the beginning of the treatment, followed by salicylates, such as sodium salicylate, 30 to 60 grains daily. In idiopathic and gonorrhœal cases salicylates are also indicated. Morphine may be necessary to quiet pain.

Local Treatment.—Dry or moist heat should be applied. In severe cases leeches may be placed near the outer canthus. Atropine is most essential (1 per cent. solution), and should be dropped into the eye three to six times a day, depending on the severity of the attack and the ease with which the pupil is dilated. A local toxic effect is sometimes noticed in the form of conjunctivitis, swelling and redness of the lids. In this case scopolamine (1 per cent.) or duboisine (1 per cent.) may be substituted. Atropine also sometimes produces a granular conjunctivitis resembling trachoma (papillary conjunctivitis). Disagreeable effects from absorption are at times noticed—dryness of the tongue and constitutional effects, as flushed face, dizziness, and rapid pulse. Small doses of morphine and pilocarpine are then indicated.

Chronic Plastic Iritis.—Iritis may assume the chronic form, most commonly in elderly rheumatic subjects. The exacerbations are frequent, but usually not severe; however, each attack thickens the pupillary membrane, and finally leads to occlusion, glaucoma, and loss of the eye. Under this head may be included the iritis of sympathetic ophthalmia (page 125).

Treatment.—Chronic iritis is best treated by atropine, antirheumatic remedies, and by making an iridectomy.

SEROUS IRITIS.

Synonyms. — Keratitis punctata posterior: Descemitis: Aquocapsulitis.

Definition and Symptoms. — [illegible] is the term used to [illegible] shows only slight ciliary [illegible] various sizes on the posterior [illegible] occupy the lower half of [illegible] base down at the [illegible] the cornea; also a [illegible] enlarged pupil. This [illegible] inflammation of the [illegible] endothelial cells of [illegible] the choroid.

Treatment. — [illegible] caution, owing to [illegible] treatment is the same as [illegible]

Purulent Iritis. — [illegible] as a purulent [illegible] following perforating [illegible] suppurative process in [illegible] purulent processes [illegible] Hypopyon [illegible]

TUBERCULOUS IRITIS.

[illegible] the eye, but may [illegible] nodules, which [illegible] of the iris. [illegible] inflammatory [illegible] deeper structures. [illegible] has been [illegible] is the [illegible] without much [illegible] small [illegible] the eye with [illegible] The disease [illegible] tuberculous [illegible]

Treatment is the same as of other forms of iritis, with proper constitutional treatment. Enucleation may be thought advisible in advancing cases to prevent general infection.

INJURIES OF THE IRIS.

Concussion of the eyeball may produce a dilatation of the pupil, sometimes irregular. This is due to paralysis of the sphincter pupillæ. It generally disappears. The pupillary edge may be torn in the form of one or more rents, or the iris may be separated at the periphery from its root, leaving a clear space (**iridodialysis**). It may be entirely torn from its attachment (**traumatic irideremia**).

Perforating wounds are usually accompanied by injury of the lens and other structures. Wounds of the cornea are often complicated by the falling in of the iris (prolapse), the treatment of which has been described (page 73). A small foreign body passing through the cornea and iris leaves a perforation which is of diagnostic value in reference to the presence of a foreign body in the eye. The greatest danger from wounds is from infection, which, if it reaches the iris, usually produces purulent iritis. When the lens is dislocated or absent, the iris, being without support, will tremble with every movement of the eye (tremulous iris, iridodonesis). In some injuries, and occasionally after extraction of cataract, with loss of vitreous, a part of the iris may be folded back upon itself, thus enlarging the pupil in that part (retroflexion).

TUMORS OF THE IRIS.

Apart from the syphilitic and tuberculous tumors of the iris already described, tumors of the iris are of rare occurrence.

Sarcoma, usually melanosarcoma, occurs at any age, but usually in middle life.

Melanoma is a benign, nonprogressive pigmented tumor.

Cysts.—Transparent or true cysts of the iris may be congenital, but are usually traumatic. They may occur from the presence of a foreign body (transplantation cyst).

Differential Diagnosis of Tumors of the Iris.—*Tubercles* occur in youth at the periphery of the iris, secondary to other tuberculous disease; bright yellow or gray color, with an occasional vessel running over the surface; associated with serous iritis; gradually infiltrate the surrounding tissues; may disappear; rupture through limbus.

Gummata.—Occur in middle life in the tertiary stage of syphilis; dark yellow color at the pupillary edge or at the periphery; break down or absorb.

Sarcomata.—Dark, usually; middle and late life; lower part of the iris; vascular; rarely burst through the cornea; progressive.

Cysts.—Transparent; usually following injury.

Congenital Defects of the Iris.—The iris is rarely entirely wanting at birth (*irideremia, aniridia*). A part of the iris from the periphery to the edge of the pupil may be congenitally absent (*coloboma of the iris*). This occurs in the inferior nasal quadrant. The pupil may be irregularly placed (*corectopia*) or multiple (*polycoria*). There may be remnants of the pupillary membrane stretching across the pupil (*persistent pupillary membrane*).

CHAPTER VIII.

DISEASES OF THE PUPIL.

PHYSIOLOGY.

Contraction.—The contraction of the pupil is caused by the action of the sphincter pupillæ. I. If **light** falls upon the retina, the pupil contracts by reflex action. The course of the impulse starting in the retina and ending in the contraction of the sphincter is as follows: From the retina it travels through the optic nerve and optic tract to the anterior corpus quadrigeminum (probably). From there the pupil-fibres pass by Meynert's fibres to the centre of the sphincter pupillæ in the third nerve nucleus. Here the reflex is started, which passes out to the eye by the third nerve and its ciliary fibres. The contraction to light of the pupil of the eye illuminated is called the *direct action*, but the other pupil also contracts at the same time. This is called the *consensual action*. It is explained by the fact that the pupillary fibres of the optic nerve probably suffer semidecussation at the chiasm as well as the visual fibres. There is also a communication between the two corpora quadrigemina and between the third nerve nuclei. II. The pupil contracts not only to light, but also to **convergence** and **accommodation**. It seems probable that the three centres for convergence, accommodation, and pupil-contraction, though independent, are stimulated simultaneously by the voluntary impulse for adjustment of the eyes for the near point.

Dilatation.—The presence of a dilator muscle is still in doubt. The elasticity of the posterior membrane and vasomotor action are probably in part active in dilatation. Dilatation is under the control of the sympathetic system. The centre is in the medulla and the course is down the spinal cord to the seventh and eighth cervical and first dorsal roots, through the cervical sympathetic, carotid, and cavernous plexus to the eye. Physiologic dilatation is produced by irritation of sensory nerves and by psychic conditions such as fright or anger.

Size of the Pupil.—There is no standard size so varied are the influences acting upon the pupil. In middle life, with moderate illumination and the accommodation at rest, it is from 4 to 4.5 mm. in diameter. In children it is larger than in the adult.

Pathology.—The state of dilated pupil is called *mydriasis;* of contracted pupil, *myosis.* Bearing in mind the physiology, it will be readily seen that *mydriasis* may be produced by (*a*) paralysis of the third nerve or those fibres going to the sphincter pupillæ, or by (*b*) stimulation of the sympathetic fibres or the dilator fibres. On the other hand, *myosis* may be produced by (*a*) paralysis of the sympathetic fibres or (*b*) irritation of the third nerve. Hence we have:

Mydriasis .	Paralytic. Spasmodic.	Myosis . .	Paralytic. Spasmodic.

Paralytic mydriasis will be produced by lesions involving the third nerve or its centre, such as tumors, locomotor ataxia, disseminate sclerosis, hemorrhages, or injuries; also by lesion of the optic nerve or tract and by paralysis of the sphincter.

Spasmodic mydriasis may be due to high intracranial pressure, spinal irritation, mental excitability.

Paralytic Myosis.—Diseases of the upper portion of the spinal cord.

Tumors or wounds involving the cervical sympathetic.

Spasmodic Myosis.—Meningitis in the early stage. Irritation of the third nerve or centre by lesions in the vicinity. As a reflex from irritation in the eye, as from a foreign body.

The so-called *Argyll-Robertson pupil* does not contract to light, but does contract to accommodation. It is principally found in locomotor ataxia.

Hippus is the alternate contraction and dilatation of abnormal amplitude.

QUESTIONS.

What is the classification of iritis?
Give the etiology, pathology, symptoms, complications, and treatment of acute plastic iritis.
Define serous iritis.
Name two forms of tuberculous iritis.
What is iridodialysis? Iridodonesis? Irideremia?
Mention the tumors of the iris. Name the congenital defects.
Describe the physiologic contraction of the pupil to light.
What are the direct and the consensual action of the pupil to light?
Explain contraction to accommodation.
Explain physiologic dilatation.
What are the pathologic states of contracted and dilated pupil?
Give examples of each.
What is the Argyll-Robertson pupil?

CHAPTER IX.

DISEASES OF THE CILIARY BODY.

CYCLITIS.

THE ciliary body is in close anatomic relations on the one side with the iris and on the other with the choroid. It is not strange that it should be rarely alone affected.

Varieties.—Cyclitis is usually divided into plastic, serous, and purulent.

ACUTE PLASTIC CYCLITIS.

Definition.—This disease is characterized by pain in the ciliary region, circumcorneal congestion, and tenderness over the ciliary body. There are usually some opacities in the anterior part of the vitreous. Glaucoma is not an uncommon complication. If the iris is involved, the symptoms of iritis are added. When the choroiditis accompanies the cyclitis, patches of exudate may be seen if the vitreous opacities will allow inspection of the fundus. The causes are the same as in acute iritis. If the disease is severe, the prognosis is bad, for permanent blocking of the pupil may occur and permanent opacities of the vitreous may result.

Chronic Plastic Cyclitis.—Definition and Symptoms.—Usually the iris is involved. There are occlusion of the pupil and formation of exudate in the vitreous back of the lens, which tends to organize and draw together the ciliary body. The periphery of the iris is retracted. Such a chronic iridocyclitis of traumatic origin may produce sympathetic ophthalmia.

Serous cyclitis is the same as the so-called serous iritis, and is described under that head.

PURULENT CYCLITIS.

Definition.—Usually the whole uveal tract is involved in the purulent inflammation—iris, ciliary body, and choroid. This condition is generally due to a perforating injury, but may occur as a metastatic condition, as in nasal disease or meningitis (see Purulent Choroiditis).

Treatment of cyclitis is the same as for iritis.

INJURIES OF THE CILIARY BODY.

The ciliary body lies behind a zone which surrounds the cornea, and is about one-fourth of an inch wide ("danger zone"). Perforating wounds in this region, followed by chronic iridocyclitis, give the conditions which cause sympathetic ophthalmia.

Wounds with or without prolapse of the ciliary body should be treated according to the rules laid down under injuries of the iris.

TUMORS OF THE CILIARY BODY.

Sarcomata, usually melanosarcoma, are of rare occurrence. **Tubercles, gummata**, and **cysts** have been reported. These may grow into the vitreous or may present in the anterior chamber at the periphery of the iris.

QUESTIONS.

Define plastic, serous, and purulent cyclitis.
What importance have wounds in the ciliary region?

CHAPTER X.

DISEASES OF THE LENS.

CATARACT.

Definition.—An opacity of the crystalline lens or its capsule.

Varieties.—Cataracts may be divided into *polar, anterior* and *posterior* (including *capsular*), *zonular, senile,* and *traumatic.* Cataracts may also be classified as *stationary* (polar and zonular) and *progressive* (senile and traumatic). When a cataract forms without known connection with other disease of the eye, it is called *primary.* If associated with glaucoma, iridocyclitis, tumors, etc., it is called *secondary.*

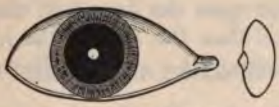
ANTERIOR POLAR CATARACT.

Synonym.—Pyramidal cataract.

Definition.—Under oblique illumination a small round dense opacity is seen at the anterior pole of the lens. This is often elevated above the level of the anterior capsule (although the capsule passes over it). It also extends somewhat into the substance of the lens. An opacity of the cornea will also generally be found near the centre.

Etiology.—Anterior polar cataract may be congenital or acquired. It usually originates from the contact of the lens with the posterior surface of the cornea after the perforation of an ulcer in infancy or *in utero*.

FIG. 18.



Anterior polar cataract, seen from the front and in section. (Nettleship.)

Symptoms.—There may be little interference with vision on account of the fact that the opacity is so near the nodal point.

Treatment is not necessary.

POSTERIOR POLAR CATARACT.

Definition and Symptoms.—May be somewhat similar to anterior polar cataract in appearance.

The **congenital form** appears as a small white round opacity, and is due to the remains of the point of contact of the hyaloid artery, which extends in fœtal life from the optic nerve through the vitreous to the posterior surface of the lens. A minute dot is very common on the posterior capsule in normal eyes, and is of similar origin. An **acquired form** is associated with intraocular disease. Cataracts beginning in the posterior cortex or upon the posterior capsule are often found associated with choroidal disease, and may clear.

ZONULAR CATARACT.

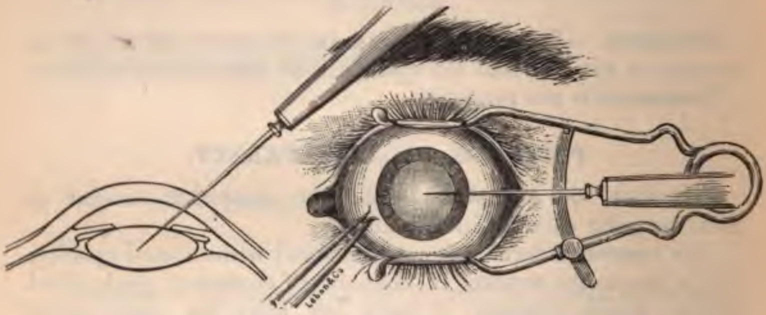
Definition.—Zonular or lamellar cataract consists of one or more opaque zones, which surround a clear nucleus and leave an outside or cortical zone clear.

Symptoms.—By oblique illumination the opacity may be seen to be lamellar in structure, often with striæ running out into the clear cortex. The extent and density of the opacity are subject to considerable variation. These cataracts are, almost without exception, stationary. Congenital cataract may also show complete opacity of the lens. A punctate and a stellate form about the nucleus exist.

Etiology.—Generally congenital; sometimes in children who have suffered from convulsions in infancy and in rachitis. There is an hereditary tendency.

Treatment depends upon the extent of the opacity. If the central opacity is less than the size of the medium pupil, and if atropine, by dilating the pupil, allows sufficient vision through the clear cortex thus exposed, an iridectomy to produce artificial pupil will be the proper treatment. The advantages of this method of treatment are that the accommodation is left intact, and no glasses need necessarily be worn. If the opacity is too extensive to obtain clear vision through an artificial pupil, the lens should be removed by absorption. The **operation for absorption by needling** (discission) is performed as follows: The pupil is dilated by atropine. The patient, if too young for self-control, should be put under a general anæsthetic. The cornea should be entered by a knife-needle which has a short, narrow blade and a long shank. A horizontal incision is then made through the anterior capsule

FIG. 19.



Discission of cataract. (Juler.)

and somewhat into the lens substance. It should be about 4 mm. in length. A vertical cross-incision may also be made if desired. The aqueous humor entering into the lens substance swells and gradually dissolves it. If the swelling is too rapid, glaucoma may ensue, in which case the lens must be let out through a corneal incision. A second and sometimes a third needling is necessary before the lens is completely absorbed. A strong convex lens must then be worn to replace the crystalline lens. Iritis may rarely complicate the operation.

SENILE CATARACT.

Etiology.—Although cataracts under this category may occur at an earlier period, the vast majority are found after fifty years of age. They are most frequent among the aged; in fact, elderly persons are likely to show some, practically stationary, opacities at the periphery of the lens. There are usually no causes to assign for senile cataracts. They appear equally in all conditions of life, although constitutional diseases, such as diabetes and tendencies toward sclerotic changes, are known to favor their development.

Pathology.—Between the ages of thirty and forty the lens begins to harden at its centre, forming what is known as the nucleus. The size of the nucleus increases with age. Cataracts are produced during the process of nucleus-formation by the irregular shrinking of the fibres and the collection of fluid within spaces thus formed. Degeneration of the fibres and coagulation of the fluid follow, producing opacities. The choroid, especially at its periphery, is at times found affected during the formation of cataract.

Subjective Symptoms.—The patient complains of blurred vision, flashes and streaks of light, dark spots, and double or multiple vision. There is never any pain directly due to cataract. There is sometimes eye-strain, due to imperfect sight. Sometimes the first subjective symptom is the ability to read without glasses (second sight). This is due to the increased refracting power of the lens from swelling.

Objective Symptoms.—I. In the early stages (*incipient cata-*

ract) an opacity may be found at the centre of the lens (*nuclear*), or radiating spiculæ may be seen in the cortex (*cortical*), or again a homogeneous or mottled opacity may appear throughout the whole lens. These changes may be best made out with the dilated pupil by oblique illumination, in which case the opacities appear white, or by the ophthalmoscope, in which case they appear black against the red reflex from the fundus. In elderly persons a brownish-red appearance without decided opacity may often be made out, especially in the centre of the lens. This is due to sclerosis (sclerosed cataract). Such a lens may appear almost black when extracted (cataracta nigra). II. As the ripening process advances (*immature cataract*), the lens becomes more extensively opaque and at the same time increases in size (*cataracta tumefacta*). This swelling of the lens is manifest in the decreased depth of the anterior chamber—*i. e.*, the iris is pushed toward the cornea. The reflex from the fundus is gradually lost and the opacity becomes quite evident in daylight. At times it has a streaked, glistening appearance (asbestiform). III. The cataract then gradually shrinks to its normal or somewhat less than normal size. It is fully opaque and "ripe" (*mature cataract*). A sclerosed cataract never becomes opaque, but translucent, and vision is never entirely lost. IV. If allowed to remain, the cortex slowly softens (*hypermature cataract*) and may become fluid, leaving the hard nucleus to float about (*Morgagnian cataract*), or the lens may become flat (disciform) or calcify.

Course.—The progress of senile cataracts is slow. A number of years usually pass before maturity is reached. They may become stationary at any time. They probably never actually improve, although a few such instances have been reported. The rapidity of progress is usually difficult to estimate by one examination, but in general sharply outlined cortical and well-defined punctate opacities are stationary. Diffused blurred opacities are progressive. In senile cataract both eyes are affected sooner or later, although it often happens that one eye may become fully mature before the other eye is materially changed.

Prognosis.—The points to be noticed before expressing an opinion as to the suitableness of a cataract for operation are as follows: The eye should be free from evidence of disease as far as one is able by external examination to exclude it—*i. e.*, dacryocystitis, conjunctivitis, corneal affections, signs of iritis, such as synechiæ (cataracta accreta). The anterior chamber should be of normal depth. The pupil should react to light. There should be a homogeneous white or gray opacity immediately back of the pupil, with no shadow from the edge of the pupil except in cases of sclerosis already mentioned. A candle carried on all sides of the patient while the eye is fixed, should be properly located by him (projection good). The tension of the eyeball should be normal.

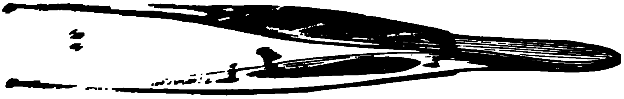
If the above examination of the eye prove satisfactory, the cataract is ripe, and in all probability vision, after operation, will be good. 95 per cent. of success is an average showing. It is of some importance to inquire of the patient as to the condition of the vision before the cataract appeared and if the eye were injured.

Treatment.—There is no control over the progress of lens opacities. Massage and electricity within justifiable limits have no appreciable effect. If both eyes are equally advanced and the vision considerably reduced, artificial ripening may be resorted to. The safest method consists in making a small opening into the anterior chamber and gently stroking the anterior surface of the lens with a spatula which is introduced through the opening. Sometimes an iridectomy (preliminary) will hasten the ripening process.

If a senile cataract has proved suitable for operation by the method of examination explained, it can be removed only by extraction, which is performed as follows:

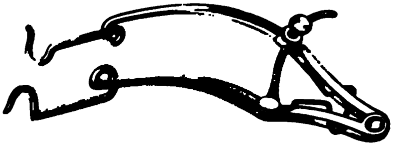
Combined and Simple Extraction.—The skin about the eye should be washed with soap and water, and the lids, especially at the roots of the lashes, thoroughly cleansed. Cocaine hydrochlorate (4 per cent.) or holocaine hydrochloride (1 per cent.) is instilled two or three times at intervals of five minutes. Before beginning the operation, the eye is flushed with normal salt solution, bichloride of mercury (1 : 5000), or boric acid solution (3 per cent.). The patient should lie on the back, if possible in bed, and the daylight or artificial illumination arranged to illuminate the eye fully. The operator should stand back of the patient's head. The lids are kept apart by *inserting a speculum*, and the conjunctiva just below the cornea is firmly seized with the *fixation forceps* held in the left hand (when operating on the right eye). The *cataract knife* (Graefe), with the edge upward, is introduced at the juncture of the cornea and the sclera on the temporal side a little above the horizontal meridian of the cornea. It is passed across the anterior chamber in front of the iris and pupil, and brought out at a corresponding point on the opposite side (counter-puncture). As soon as the point is seen outside the eye, the knife should be made to cut upward as it advances, always keeping the incision in one plane at the sclerocorneal junction.

Fig. 20.



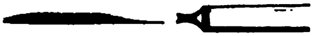
Fixation forceps.

Fig. 21.



Eye speculum.

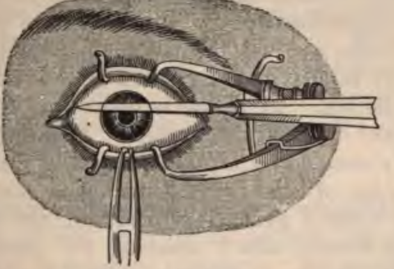
Fig. 22.



Cataract knife.

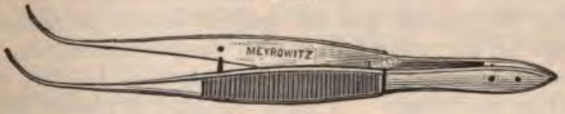
One or two movements of the knife forward and backward will finish the section. Such a section will include nearly one-half the periphery of the cornea, but should be somewhat smaller if an iridectomy is to be made. Some operators prefer to finish the section more in the cornea by turning the blade forward; others, more in the sclera, forming a flap of conjunctiva, by turning the knife back toward the equator of the eye.

FIG. 23.



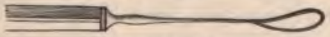
The corneal section in cataract extraction. Puncture and counterpuncture have been made. The section will pass in its whole extent exactly through the transparent margin of the cornea, the knife remaining in the same plane throughout. Slightly modified from de Schweinitz. (Ellett.)

FIG. 24.



Iris forceps.

FIG. 25.



Lens scoop.

The next step (which is omitted in the so-called *simple extraction*) is the iridectomy. While an assistant holds the fixation forceps, the operator, entering through the wound with closed *iris forceps*, seizes the iris near the pupillary edge and pulls it out through the wound. A piece is then cut off with the scissors, close to the eye. When the iridectomy is performed, the operation is called the *combined method*.

The next step (which follows the section in the simple operation) is the capsulotomy. The *capsulotome* is entered from the temporal side, and the capsule is opened by a number of scratches either in the pupillary area or above the edge of the pupil under the iris. If properly done, the lens will be seen to come forward toward the cornea. The fixation forceps is then carefully removed and, with a spoon, pressure is made at the lower edge of the cornea toward the centre of the eyeball. If the force is gradually increased, the lens will enter and open the wound, slowly dilating the pupil, and will be delivered. When the equator of the lens has passed the wound, the pressure should be somewhat relaxed and the spoon should be passed over the cornea, following the lens out. The speculum is then carefully removed. Some operators prefer to remove the speculum and extract the lens by pressing the lower lid against the eyeball at the lower edge of the cornea. Cortical matter remaining behind should be worked out by using the lower lid against the cornea. The pupil should be made round with a spatula by freeing the iris from the wound, and the spatula should be passed along the edge of the wound to assure proper apposition. If an iridectomy has been made, the cut edges of the iris must be freed from the wound. A light bandage is placed over both eyes and the patient kept absolutely quiet on the back in bed. No injury should be allowed to happen to the eye by any movement of the head or hands. On the following day it is customary to inspect the eye, and if, in case of simple extraction, the iris is found caught in the wound (prolapse), it should be drawn out with the iris forceps and cut off. A drop of atropine (1 per cent.) should be instilled at each daily dressing. Bandages may be left off the other eye in four or five days, and from the one operated on about the seventh

The patients are not allowed to leave the hospital two weeks.

Accidents and complications liable to occur are improper place and size of the incision, difficult delivery due to adhesive lens, small incision, or imperfect opening of the capsule. There may be rupture of the suspensory ligament and prolapse of vitreous due to too large incision, too much pressure, patient squeezing the lids, or to frail suspensory ligament. In the after-treatment there may occur striated keratitis (usually harmless), prolapse of iris, iritis beginning generally after the third day, iridocyclitis resulting in destruction of the eye, suppuration of the wound, intraocular hemorrhage, or injury from hitting the eye.

After-cataract.—The capsule out of which the lens is taken in many cases is left as a more or less opaque membrane across the pupil (secondary cataract). At any time after the eye has become free from congestion or irritation this may be cut with a knife-needle (discission-knife) by making a large crucial incision. Two needles entered on opposite sides of the cornea are sometimes employed. If there are tough bands, they may be cut with small scissors, as those of de Wecker. Glaucoma rarely occurs after these operations. The loss of the refracting power of the eye by removing the lens—a condition known as *aphakia*—must be made good by wearing a strong convex lens (cataract glass) in order to obtain the best vision. Usually about +10 D. is required. Often some astigmatism is also present.

Traumatic Cataract.—If the lens capsule is opened, the aqueous enters and produces swelling and opacity of the lens. Traumatic cataract may also occur from contusion without perforating injury. In this case the suspensory ligament is generally ruptured, or, in rare cases, the capsule. Rupture of the capsule in a patient under thirty-five may be followed by gradual and complete absorption of the cataract. In injured and diseased eyes the lens may calcify.

Treatment.—Traumatic cataracts should be treated according to the principles already laid down, by absorption or extraction, depending upon the age of the patient.

DISLOCATION OF THE LENS

Definition.—The lens may be displaced partially subluxation or completely luxation from its position behind the iris by rupture of the suspensory ligament by which it is attached at the capsule at its equator to the ciliary body and ciliary processes.

Etiology.—Congenital, secondary to pathologic changes, or traumatic.

I. Dislocation may be backward, upward, downward, or to the side in the vitreous chamber. The dislocated edge may often be seen with the ophthalmoscope as a curved black line in the pupil. Every movement of the eye will shake the lens and the iris will tremble iridodonesis or tremulous iris.

II. The lens may be dislocated partly through the pupil or entirely into the anterior chamber. In the latter case when it is clear it is difficult to see. Glaucoma usually supervenes.

III. The lens may be dislocated through a wound in the sclera, and lie under the conjunctiva. A dislocated lens is likely to become cataractous.

Treatment.—If inflammation or glaucoma occur, the lens should be removed after the usual cataract incision, either by the use of a fenestrated spoon or by pressure.

CONGENITAL AFFECTIONS.

Besides those mentioned the following occur:

Lenticonus Posterior.—A rare congenital affection consisting of a bulging of the centre of the posterior surface of the lens. Lenticonus anterior is extremely rare.

Coloboma is a rare condition, in which part of the lens is absent.

QUESTIONS.

Define and give classification of cataract.
Explain anterior and posterior cataract.
Define and give rules for the treatment of zonular cataract.
What are the etiology and pathology of senile cataract?
Describe four stages in the development of senile cataract.
Describe the examination of the eye for the determination of fitness for operation.

What is artificial ripening?
Describe the operation for extraction of cataract.
What is an after-cataract?
Describe traumatic cataract.
In what directions may the lens be dislocated?
Mention congenital affections of the lens.

CHAPTER XI.

DISEASES OF THE VITREOUS.

THE vitreous humor should be perfectly clear to all objective methods of examination, but subjectively floating opacities (**muscæ volitantes**) may always be seen if properly searched for. They are the source of considerable annoyance to nervous individuals. They are shadows thrown on the retina by the vitreous cells.

OPACITIES OF THE VITREOUS.

Opacities **vary** in form, character, and origin. They interfere more or less with vision. They may be either fixed or floating. In the latter case the vitreous is fluid in consistence (**synchysis**). The ophthalmoscope is used in the examination of the vitreous.

Fixed Opacities.—One is the remains of the hyaloid artery in the canal of Cloquet. This appears as a band extending at varying distances from the optic nerve into the vitreous, even to the posterior capsule of the lens. Other fixed opacities are bands or membranes of connective tissue, which may be congenital or the result of organization of inflammatory exudate.

Floating opacities may be dust-like and fill the whole vitreous (characteristic of syphilis), or large masses, bands or membranes secondary to inflammation of the ciliary body, choroid, or retina, to hemorrhage, injury, or to degeneration of the vitreous. Degenerative changes occur in high myopia, old age, exhaustion from depressing disease, menstrual disorders, systemic disturbances, or may be idiopathic.

Synchysis scintillans is a peculiar condition in which the vitreous is filled with numerous scales which reflect the light as brilliant floating spots. These are cholesterin crystals.

Treatment.—If any constitutional disease may be made out, it should be treated. If the cause of the vitreous opacities is inflammation in the adjacent tissues, attention should be given to this.

SUPPURATIVE INFLAMMATION OF THE VITREOUS.

Synonym.—Purulent hyalitis.

Etiology.—Pus in the vitreous may be due to infection from perforating wounds, or may be of metastatic origin from meningitis, infectious diseases, or from ear or nose. It may follow inflammations of the uveal tract, or occur spontaneously in debilitating diseases.

Symptoms.—The cornea, aqueous, and lens are clear, but a yellow reflex is obtained back of the lens. A few posterior synechiæ may be found. If the disease is advanced, the eye will be soft and the periphery of the iris will be drawn back by traction on the ciliary processes from within. Such a condition may be mistaken for glioma of the retina, and is sometimes called **pseudoglioma** (see Glioma of the Retina). When the process is acute and severe, it is known as **panophthalmitis**, or **abscess of the eye**, in which case the whole eye and surrounding orbital tissue are involved.

Treatment.—If the inflammatory conditions subside, it is not necessary to perform enucleation for pseudoglioma. In panophthalmitis, hot fomentations and incision into the eye to evacuate pus are indicated. Enucleation has been followed by meningitis.

QUESTIONS.

What are muscæ volitantes?
What are the forms of opacities of the vitreous?
Define synchysis scintillans.
Describe suppuration of the vitreous and give treatment.
What is pseudoglioma?

CHAPTER XII.

DISEASES OF THE RETINA.

Anæmia and **Hyperæmia** of the retina occasionally occur as the result of local or general conditions, but the variations may be considerable within physiologic limits.

Retinitis.—Simple, albuminuric, syphilitic, and pigmented varieties of retinitis occur.

Retinitis is often associated with choroiditis (chororetinitis) or with inflammation of the optic nerve (neuroretinitis).

SIMPLE RETINITIS.

Etiology.—The causes are often obscure. There is commonly some constitutional disturbance—*e. g.*, arteriosclerosis, anæmia, leukæmia, malaria, etc. Cases may be due to albuminuria, syphilis, or diabetes; but such causes generally produce characteristic appearances, which will be described under separate heads.

Subjective Symptoms.—The patient complains of impaired vision and blurs in the field of vision and flashes of light. There may be photophobia and distortion of objects (metamorphopsia).

Objective Symptoms.—There may be found only slight disturbance of the retina, such as dilated veins, tortuous vessels, and a few hemorrhages; or, in more marked cases, a cloudy fundus, dilated and distorted vessels buried in the swollen retina, with numerous flame-like hemorrhages, and the outline of the nerve is blurred. The disease may be unilateral or bilateral. The duration is usually several months.

Treatment.—The eyes should be protected from light and strain. Mercury, or potassium iodide, sodium salicylate, and diaphoretics may be given.

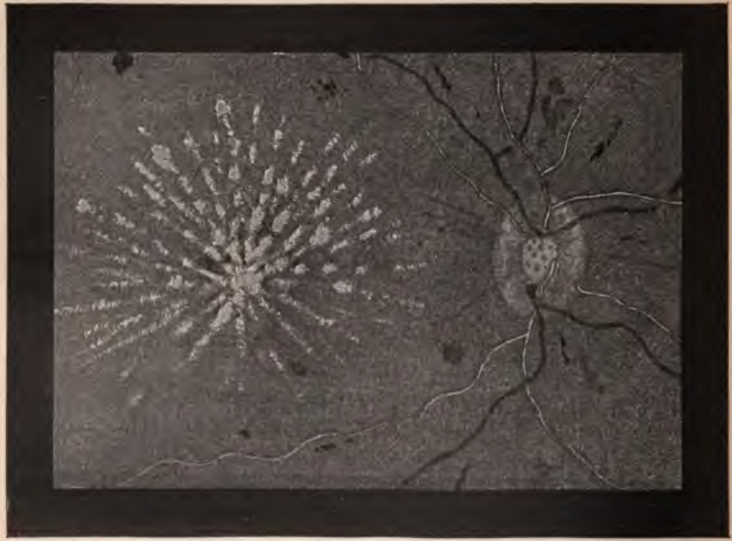
Hemorrhagic Retinitis.—A form of simple retinitis in which hemorrhages are the most striking feature. It is most common in elderly people with apoplectic tendencies due to arteriosclerosis. Thrombosis of the retinal veins may occasion hemorrhages with dilated veins. At times hemorrhage occurs between the retina and vitreous (subhyaloid).

ALBUMINURIC RETINITIS.

Definition.—A distinct type of retinitis accompanying acute or advanced chronic nephritis. Usually bilateral.

Etiology.—The cause is nephritis, usually chronic interstitial. It also occurs in Bright's disease of pregnancy and in acute nephritis. Diabetes is the cause of a somewhat similar condition in the retina.

FIG. 26.



Albuminuric retinitis. Granular kidney. Note hard-edged "asterisk" exudation at macula, and the punctate and linear hemorrhages. (Posey and Wright.)

The only **subjective symptom** is interference with vision. This is sometimes surprisingly slight in cases showing marked fundus changes. Patients with nephritis are subject to attacks of temporary blindness of uræmic origin, with or without retinitis. By **ophthalmoscopy** there may appear the signs of simple retinitis—swelling, tortuous vessels, hemorrhages, and, in addition, shining white patches scattered through the fundus, and a peculiar arrangement of glistening white dots around the macula. This is a stellate figure formed by radiating lines.

Pathology.—The white patches are due to fatty degeneration of the retinal elements and to exudate.

Prognosis.—Retinitis in chronic nephritis is a late manifestation, and the patient is not likely to live more than two years after the appearance of the eye lesion. Temporary improvement may occur, especially when the disease assumes an acute or inflammatory exudative form.

Treatment.—No local treatment is of use. When the disease appears in pregnancy, the question of producing premature labor is a grave one. If the retinitis is marked and occurs before the seventh month, it may be wise to induce labor. If after the seventh month, and mild, it is better to wait.

SYPHILITIC RETINITIS.

Etiology.—Occurs in the second stage of acquired syphilis and in the congenital form.

Subjective symptoms are the same as for other forms of retinitis.

Objective Symptoms.—The ophthalmoscope shows dust-like opacities in the vitreous—a peculiar bluish-gray haze over the retina, about the disk and macula, and streaks of white exudate along the vessels.

Course.—The course is chronic, leading to choroiditis and atrophy of the optic nerve.

Treatment.—Antisyphilitic. Should be energetic, and if begun early may be successful.

RETINITIS PIGMENTOSA.

Definition.—A disease characterized by a prolonged course, beginning in youth and prolonged for years. There is loss of vision, especially in subdued light, as in the twilight or evening (night blindness, nyctalopia). There is a peculiar arrangement of the retinal pigment into masses of irregular shape, mostly with branching projections. These appear first at the periphery, later approach the nerve. The field of vision becomes gradually narrowed. The nerve and retina become atrophic, and the bloodvessels of the retina much reduced in calibre. There is a strong hereditary tendency, and consanguinity in parents is an element.

EMBOLISM OF THE CENTRAL ARTERY OF THE RETINA.

Definition.—Plugging of the central artery, or more rarely a single branch.

Etiology.—There may be heart lesion or obliterating endarteritis of the retinal vessels.

Symptoms.—There is sudden blindness in one eye without pain or other symptoms. The retina in a short time begins to assume a foggy appearance (œdema), especially near the centre of the fundus. A cherry-red spot is found at the macula. The arteries are small and the veins contain little blood. Later the circulation may be restored, the blood returning at times in broken columns. Atrophy of the retina and nerve usually results. Occasionally central vision is preserved, which is due to the existence of a branch of the central artery coming off back of the location of the embolus (or of a branch from the ciliary arteries), supplying the macular region.

Treatment.—Massage is recommended.

DETACHMENT OF THE RETINA.

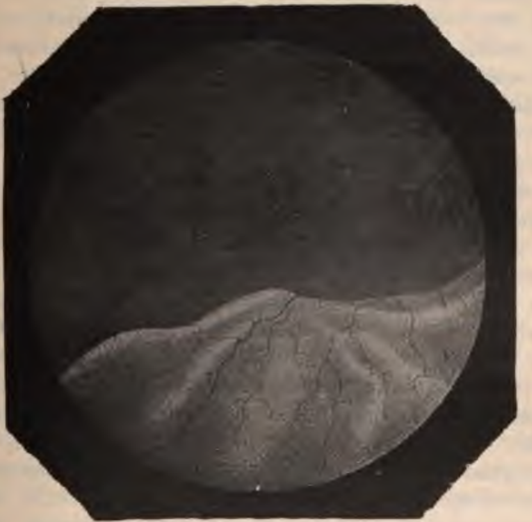
Synonym.—Ablatio retinæ.

Definition.—Separation of the retina from the choroid, leaving behind the layer of retinal pigment.

Etiology.—This may occur from the extravasation of blood or serum, or from the presence of exudate or new growth. It may also occur from the traction of bands of connective tissue in the vitreous. The ordinary form occurs as a complication of myopia of high degree. Traumatism is the next most frequent cause.

Symptoms.—The patient complains of poor vision and defect in the field of vision corresponding to the detachment. The ophthalmoscope often reveals floating opacities in the vitreous. The detached retina appears as a wavy grayish or greenish-white membrane, over which the very dark-red retinal bloodvessels run in tortuous course. The retina usually floats about with the movements of the eye. A tear is sometimes seen. The tension of the eye is reduced.

FIG. 27.



Detachment of the retina. (Jaeger.)

Prognosis.—These cases, especially if complicating myopia, get worse, and all vision is lost. The cases occurring as the result of traumatism are the most favorable, but the prognosis is always bad.

Treatment.—In recent cases rest in bed for a number of weeks should be ordered; also hypodermatic injections of pilocarpine (gr. $\frac{1}{10}$), to produce sweating, every day or two. Puncture of the sclera over the detachment and subconjunctival injections are of questionable value.

GLIOMA OF THE RETINA.

Definition.—A malignant intraocular tumor occurring in early childhood or infancy, commonly before three years of age.

Pathology.—Glioma springs from the granular layer of the retina, grows underneath the retina (exophytum), or on top of it in the vitreous (endophytum). It consists of bloodvessels, small round cells, and cells with protoplasmic processes in scant stroma.

Symptoms.—In the *first stage* there appears a shining white or yellowish reflex from the interior of the eye (formerly called "amaurotic cat's eye"). The eye is blind. A few bloodvessels may be seen on the mass in the vitreous. In the *second stage* (glaucomatous) the eye becomes hard by the growth of the tumor filling the interior. It is painful and somewhat congested. *Third stage.* The new growth bursts through the eye and extends either backward into the orbit or forward toward the outside. *Fourth stage.* Metastatic growths appear in other organs. The other eye may be affected, and the child dies of cerebral complications or from exhaustion. Other children in the same family are sometimes affected.

Differential Diagnosis.—The condition may be mistaken for purulent choroiditis (**pseudoglioma**).

Glioma occurs in early childhood, with no history of injury or meningitis. Tumor often well defined, with rest of vitreous clear. Anterior chamber shallow and tension increased.

Pseudoglioma may occur at any age; follows injury or meningitis. Vitreous immediately and wholly filled with yellowish mass. Inflammatory signs appear early. The iris is bulging at the pupillary edge, but retracted at the periphery. Tension is minus.

Treatment.—The eye should be enucleated at the earliest moment.

OPAQUE NERVE-FIBRES.

Definition.—A congenital anomaly showing flame-like, glistening, white patches extending from the optic nerve into the retina. They are formed of nerve-fibres which have retained their medullary sheaths, normally lost as the fibres pass through the lamina cribrosa into the eye.

INJURIES OF THE RETINA.

Beside the diseases already mentioned as due to traumatism, contusions and wounds may occur. **Commotio retinæ** (œdema of the retina) arises from contusions and shows defective vision and gray infiltration of the retina, especially in the macular region.

RARER FORMS OF RETINAL DISEASE.

Retinitis Proliferans.—Characterized by masses of connective tissue in the vitreous, which contain bloodvessels. Hemorrhages are undoubtedly the original lesion.

Retinitis Circinata.—Presenting a white circle of exudate concentric with the macula.

Retinitis Striata.—Showing white streaks of fibrous tissue in the retina.

Angioid Streaks.—Black or brown striæ, in deeper layer; probably due to hemorrhages.

Snow Blindness.—From exposure to brilliant light, such as from snow or electric light. There may be central scotoma, macular changes, and retinitis.

Amaurotic Family Idiocy.—Symmetric changes at the macula in infancy. A peculiar condition, showing a hazy area in the macular region, with a red spot in the centre. The disease is probably due to degeneration of ganglion-cells of the retina, following arrest of development of the nervous system. There is a strong hereditary influence. Jews are more prone to the disease (Jacobi). The children all die within a year or two.

AMBLYOPIA OR FUNCTIONAL DISEASE OF THE RETINA.

In the ordinary acceptance of the term amblyopia is used to signify a defect in vision without discoverable lesion or refractive error. Amaurosis is a name which has fallen into disuse, but signifies total blindness from unknown causes.

The forms of amblyopia are (I.) congenital, (II.) hysterical, (III.) simulated, and (IV.) toxic.

I. Congenital Amblyopia.—Commonly associated with errors of refraction, especially hypermetropia and astigmatism. In convergent strabismus the squinting eye is likely to be amblyopic. It is commonly believed that this is from nonuse (amblyopia ex anopsia).

II. Hysterical Amblyopia.—Generally unilateral. Blindness may be partial or total. The field of vision is contracted, especially after repeated examinations. The fields for colors are reversed as to their order in point of size.

III. Simulated Amblyopia.—Malingering is not unknown, and is attempted for various reasons. Generally blindness in one eye only is feigned. When a prism is placed over one eye, such patients will often acknowledge diplopia, and will find difficulty in walking, etc.

IV. Toxic Amblyopia.—Under this head may be classed amblyopia occurring in uræmia, malaria, and poisoning with drugs, such as quinine, tobacco, and alcohol. Lesions, especially of the nerve, are often developed.

QUESTIONS.

Mention the varieties of retinitis.

Give the etiology, symptoms, and treatment of simple retinitis.

What is albuminuric retinitis? Give the etiology, symptoms, pathology, prognosis, and treatment.

Describe syphilitic retinitis.

Define retinitis pigmentosa.

Explain embolism of the central artery of the retina.

Give the etiology, symptoms, prognosis, and treatment of detachment of the retina.

Give the four stages of glioma of the retina.

Give the differential diagnosis between glioma and pseudoglioma.

What are opaque nerve-fibres?

What diseases may be produced by traumatism?

What constitutional diseases produce affections of the retina?

Describe the four forms of amblyopia.

CHAPTER XIII.

DISEASES OF THE CHOROID.

THE choroid is the vascular tunic of the eye and liable to inflammation. Its intimate connection with the retina makes their simultaneous involvement of common occurrence, although the entirely separate blood-supply renders disease of each distinctive.

Varieties.—Exudative choroiditis, serous choroiditis, suppurative choroiditis, and sclerochoroiditis posterior.

EXUDATIVE CHOROIDITIS.

Definition.—A disease characterized by localized patches of plastic inflammation, later producing atrophic areas.

Pathology.—The exudate is a collection of round cells in the choroid and in the outer layers of the retina. Organization of this exudate causes atrophy and disturbance in the pigment.

Etiology.—Syphilis (congenital or acquired) is the most common cause, also general disorders of nutrition, and it may be idiopathic. Tuberculous deposits occur very rarely.

Varieties.—*Central.* Occurs not infrequently in myopia, syphilis, and in elderly persons as a senile change. *Disseminate.* Essentially chronic, characterized by scattered patches. *Diffuse.* Always accompanied by involvement of the retina and called chorioretinitis. Due to syphilis. *Isolated.* Some cases are characterized by isolated patches of exudate occurring in people as result of overexertion or idiopathic. Not due to syphilis. Runs a comparatively short course.

Symptoms.—Gradual loss of sight is complained of, although the vision is sometimes good, with extensive changes. The field of vision may be contracted, and there may be scotomata. When recent, the diseased areas appear as irregular hazy, white or yellowish patches. Isolated hemorrhages may occur. In the stage of atrophy there are masses of pigment or white patches with or without rings of pigment about them. Opacities of the vitreous and lens or atrophy of the optic nerve may complicate the case.

Treatment.—Mercury, either internally or by inunctions, is beneficial even in nonspecific cases. Iodide of potassium in full doses or large doses of salicylate of sodium are of value.

Serous Choroiditis. (See Serous Iritis.)

SUPPURATIVE CHOROIDITIS.

Definition.—This condition is practically the same as described under suppurative inflammation of the vitreous. On rare occasions in the course of infectious or wasting diseases the beginning of the suppurative process may be seen as foci of infection in the choroid. These quickly involve the whole interior of the eye in purulent inflammation.

Treatment.—Unless the case may be aborted, which is practically impossible, the eye is lost.

SCLEROCHOROIDITIS POSTERIOR.

Definition.—Consists in a slow process of atrophy of the choroid surrounding the optic nerve, especially on the side toward the macula. It complicates myopia, and is associated with bulging of the sclera in this place (posterior staphyloma). See page 132.

TUMORS OF THE CHOROID.

Sarcoma, gumma, tubercle. and secondary carcinoma are of rare occurrence.

SARCOMA OF THE CHOROID.

Pathology.—Melanosarcoma is the most common form.

Symptoms.—*First Stage.*—The patient complains of blurred vision. A tumor will be seen in some part of the fundus. Over it other vessels beside those of the retina will be seen. The retina is often detached on one side.

Second Stage.—The eyeball becomes hard and painful, and the vision is lost.

Third Stage.—The tumor involves the surrounding parts. It ruptures through the globe or extends backward through the optic nerve.

Fourth Stage.—Metastatic growths appear.

Diagnosis.—A tumor showing vessels beside those of the retina, with increase of tension.

Prognosis.—Unless seen in the early stage, the prognosis is decidedly bad.

Treatment.—Immediate enucleation, with the optic nerve cut well back.

INJURIES OF THE CHOROID.

Beside the direct laceration of the choroid in perforating injuries, it may be *ruptured* by contusions. Such a condition shows a curved white line of exposed sclera bordered with pigment usually concentric with the optic nerve.

Treatment.—None is possible.

CHOROIDAL HEMORRHAGE.

Choroidal hemorrhage may occur from injury or from sudden relief of pressure in operations which open the eyeball.

CONGENITAL DEFECTS OF THE CHOROID.

Coloboma is a defect in development owing to imperfect closure of the fœtal cleft. It appears as a white area of exposed sclera, stretching usually from the optic nerve toward the ciliary body and iris, which may be involved in the same way. There are often irregularities in the surface of the area, which is bordered with more or less pigment. An uncommon form of coloboma is confined to the macula.

Albinism of the eye is a condition of absence of pigment.

QUESTIONS.

Name the varieties of choroiditis.
Name the varieties of exudative choroiditis.
Describe four stages of sarcoma of the choroid.
Describe rupture of the choroid and congenital defects.

CHAPTER XIV.

DISEASES OF THE OPTIC NERVE.

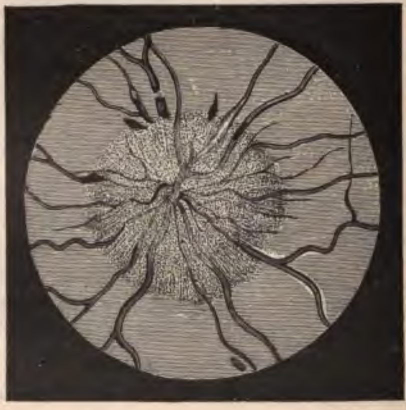
THE diagnosis of **anæmia or hyperæmia of the optic nerve** should be made with care, in view of variations within physiologic limits. If the optic nerve is inflamed at its entrance into the eye, the condition is called **optic neuritis, intraocular neuritis,** or **papillitis.** If the process is confined to the nerve behind the eyeball, it is known as **retrobulbar neuritis.**

OPTIC NEURITIS.

Definition.—An inflammation of the head of the optic nerve, characterized by congestion and swelling of the optic disk.

Etiology.—Papillitis occurs in brain tumors, generally in the form known as "*choked disk.*" It is also caused by syphilis, nephritis, rheumatism, anæmia, diseases of the vascular system, infectious diseases, and poisons, as well as diseases of the orbit

FIG. 28.



Ophthalmoscopic appearance of severe recent papillitis. Several elongated patches of blood near border of disk. (After Hughlings Jackson.)

and adjacent sinuses. Tuberculous and other forms of meningitis are the cause of *descending neuritis.*

Pathology.—There is infiltration of white cells. The pathology of "choked disk" is yet unsettled, but this condition seems to be due to increased intracranial pressure, with distention of the optic nerve sheath.

Subjective Symptoms.—The patient complains of defective vision, which is, however, independent of the apparent severity of the lesion, for marked cases may retain excellent vision.

Objective Symptoms.—There are no external signs. With the ophthalmoscope the optic disk is congested or of whitish color. The edges are blurred and streaked. There is more or less swelling of the disk. The veins are distended and tortuous and the arteries small. There may be hemorrhages. The field of vision is usually defective. The neighboring retina is always involved to some extent, but there may be general retinitis (neuroretinitis). The disease is usually bilateral. In "choked disk" there is great swelling of the nerve (œdema), dilatation of vessels, and hemorrhages.

Course.—The disease lasts for months. The inflammation may subside without after-effects, or the nerve may pass into a condition of atrophy (postneuritic).

Treatment.—The treatment consists in giving attention to the cause, sweating with pilocarpine, potassium iodide, sodium salicylate, and rest in bed.

ACUTE RETROBULBAR NEURITIS.

Definition.—An inflammation of the orbital portion of the optic nerve.

Etiology.—Syphilis, rheumatism, infectious diseases, poisons—*e. g.*, methyl alcohol—and secondary to inflammation in the adjacent tissues.

Pathology.—In most cases the inflammation is confined to the fibres which supply the macula.

Symptoms.—Rapid loss of sight, orbital pain and tenderness. There may be no intraocular evidence of disease or only appearances of moderate optic neuritis. Optic nerve atrophy, especially on the temporal side of the disk, is likely to follow with central scotoma.

Treatment.—Same as for optic neuritis.

CHRONIC RETROBULBAR NEURITIS.

Synonym. Toxic amblyopia.

Definition. A disease characterized by gradual loss of vision and atrophy of the optic disk on the temporal side.

Etiology. Nicotine, especially combined with alcohol, is the most common cause. Lead, arsenic, bisulphide of carbon, and other poisons may also produce it. It occurs in middle and late life.

Pathology. The lesion is a chronic interstitial inflammation of the macular fibres of the nerve, which occupy the temporal [illegible] the nerve at the optic disk, but pass into its centre [illegible] back in the orbit.

Symptoms. The patients find their sight gradually failing. There is diminution in central vision (central relative scotoma) and loss of color perception in a small area about the fixation point (central color scotoma). The ophthalmoscope shows a [illegible] pallor of the disk on the outer side and [illegible] of the retinal veins. The disease affects both eyes.

Course. It is of long duration, but never produces total blindness.

Treatment. Alcohol and tobacco should be absolutely cut off. Strychnine may be given to the physiologic limit; also potassium iodide.

ATROPHY OF THE OPTIC NERVE.

Definition. A condition of the optic nerve characterized by degeneration and shrinking of its fibres, and showing a white or gray disk.

Etiology. Atrophy may be: *a.* Primary, idiopathic, or associated with diseases of the brain or spinal cord. There is an hereditary type, which affects members of the same It begins in youth, and gradually produces total *b.* Secondary to optic neuritis or diseases of the choroid and retina and glaucoma; also after injuries and compression.

Pathology.—There is chronic interstitial inflammation, with atrophy of the nerve-fibres.

Symptoms.—The patient has no symptoms except gradual loss of sight and perhaps contraction of the field of vision. The disk varies in appearance from the slightest degree of pallor to the most intense white or gray. In advanced cases there will be noticed a depression in the centre of the disk, with sloping sides. The edges of the nerve are sharply defined, except when following optic neuritis. There are concentric or irregular contraction of the field of vision and defects in color perception.

Course is usually long.

Treatment.—If any indications for general treatment can be found, they should be followed. Strychnine in full doses, preferably by the hypodermatic method, potassium iodide, arsenic, electricity, and massage, may be tried, but are of little value.

Tumors of the Optic Nerve.—Fibroma, sarcoma, endothelioma, myxoma, tubercle, and glioma are known to occur. Hyaline bodies are found on the disk.

CONGENITAL AFFECTIONS.

Inferior Conus.—A congenital, white crescent, usually on the lower side of the nerve.

Coloboma of Optic Nerve-sheath shows a depression on the lower side of the disk, due to absence of the sheath.

QUESTIONS.

State the two forms of inflammation of the optic nerve.
Describe acute and chronic papillitis; also retrobulbar neuritis.
Give the etiology, symptoms, and treatment of atrophy of the optic nerve.
What general diseases cause affections of the optic nerve?

CHAPTER XV.

DISEASES OF THE ORBIT.

PERIOSTITIS.

Etiology.—Occurs, especially at the margin, from traumatism, syphilis, rheumatism, tuberculosis, and extension from the neighboring sinuses.

Symptoms are pain, tenderness, swelling, and perhaps abscess, with fistula and contracture, later producing cicatricial ectropion.

Treatment.—Constitutional, hot applications, and incision.

ORBITAL CELLULITIS.

Definition.—Inflammation of the cellular tissue of the orbit, usually terminating in suppuration.

Etiology.—Injuries, erysipelas, septicæmia, extensions from neighboring sinuses, and idiopathic.

Symptoms.—There may be constitutional disturbance, swelling of the lids, chemosis, exophthalmos, and perhaps panophthalmitis and meningitis.

Treatment.—Hot applications, incision into the orbit if pus has formed.

TUMORS OF THE ORBIT.

Cyst, aneurism, angioma, osteoma, sarcoma, and carcinoma have been reported.

Treatment.—Excision, with preservation of the eye if possible. The operation of Krönlein (osteoplastic) consists in removing part of the outer wall of the orbit, and gives access to the orbit. The whole contents of the orbit are sometimes removed in case of malignant disease (exenteration).

QUESTIONS.

Describe periostitis and orbital cellulitis.
Give the treatment of tumors of the orbit.

CHAPTER XVI.

DISEASES OF THE EYEBALL.

EXOPHTHALMOS.

Synonym.—Proptosis.

Definition.—A condition in which the eyeball is pushed forward from orbital inflammation, hemorrhage, tumors, or in exophthalmic goitre.

PULSATING EXOPHTHALMOS.

Definition.—Protrusion, with pulsation of the eyeball and surrounding parts. A bruit is heard above the eye. Generally due to traumatism causing communication between the carotid artery and cavernous sinus.

Treatment.—Tying the common carotid artery.

EXOPHTHALMIC GOITRE.

Synonyms.—Graves' disease, Basedow's disease.

Definition.—A disease of the nervous system in which the heart action is accelerated, thyroid gland enlarged, and the eyes prominent.

Subjective and Objective Symptoms.—The exophthalmos is partly only apparent on account of the widening of the palpebral fissure (Dalrymple's symptom). When the eyes are turned downward, the upper lid does not follow (Graefe's symptom). There is also infrequent winking (Stellwag's symptom). If the exophthalmos is extreme, the cornea suffers from exposure, since the lids can not be closed.

Treatment.—Beside general treatment, thyroidectomy and sympathectomy have been tried.

Enophthalmos is recession of the eyeball into the orbit. Usually traumatic and very rare.

Anophthalmos is absence of the eyeball. It may be congenital.

Megalophthalmos is enlarged eyeball.

Microphthalmos is a [illegible] congenital [illegible] resulting from extensive inflammation [illegible] phthisis bulbi.

BUPHTHALMOS.

Synonyms.—Hydrophthalmos; Keratoglobus; Congenital [illegible]

Definition.—A progressive enlargement of the whole eye.

Occurrence.—Begins *in utero* or in infancy.

WOUNDS OF THE EYEBALL.

[illegible] the eyeball have been described [illegible] structures of the eye.

FOREIGN BODIES WITHIN THE EYEBALL.

[illegible] of a foreign body are [illegible] danger of infection [illegible] the wound, the pres- [illegible] leads to destructive

[illegible] the wound, be- [illegible] sometimes a view of [illegible] its presence in the [illegible] magnetic needle prop- [illegible] may be used. [illegible] the eye, pain is [illegible] successfully em- [illegible]

[illegible] wood, stone, brass, [illegible] or in any [illegible] it may be [illegible] through an incision in [illegible] into the ante- [illegible] such as that employed, [illegible] smaller magnets are sometimes of service, and may even be introduced into the wound. It is well to remember that small aseptic foreign bodies may become encysted and remain innocuous for many years. Even after successful extraction, degenerative changes are likely to occur after a considerable time.

ENUCLEATION.

This operation is performed under general anæsthesia. The conjunctiva is cut all about the edge of the cornea with scissors. Each muscle is then taken on a strabismus hook and cut close to the eyeball. A strong scissors, which is curved on the flat, is passed backward close to the eyeball on the nasal side until the optic nerve is felt. This is severed with one stroke. The eye is then easily freed from remaining adhesions. After cleansing with an antiseptic (bichloride of mercury 1 : 5000) the conjunctiva should be brought together with a purse-string suture. An artificial eye may be worn in two or three weeks.

EVISCERATION (EXENTERATIO BULBI).

A substitute for enucleation, having the advantage of leaving a better stump for an artificial eye. The eye is opened by excising the cornea, and the contents are scraped out, leaving only the sclera. A hollow sphere of glass or silver is sometimes introduced (Mules' operation).

QUESTIONS.

Define pulsating exophthalmos and exophthalmic goitre.

What are the dangers of perforating wounds of the eyeball?

How may the diagnosis of the presence of foreign bodies within the eyeball be made?

Give the treatment of foreign body within the eyeball.

Describe enucleation and evisceration.

CHAPTER XVII.

GLAUCOMA.

Definition.—A disease characterized by increase of intraocular tension.

Varieties.—Acute inflammatory glaucoma, chronic inflammatory glaucoma, simple glaucoma, and secondary glaucoma.

ACUTE INFLAMMATORY GLAUCOMA.

Predisposing causes are, age over forty, arterial sclerosis, high arterial tension, and hypermetropia.

Exciting causes are physical and mental depression, insomnia, and the use of a mydriatic.

Pathology.—A current normally passes from the ciliary body, around the lens, through the pupil, anterior chamber, and ligamentum pectinati in the iris angle, into Schlemm's canal. Interference with this system of circulation, such as would be caused by blocking-up of the iris angle and cutting off the outflow, with or without increase in the secretion, will be followed by rise in intraocular tension. Upon this theory most explanations of glaucoma are founded.

Prodromal Symptoms.—For a year or two the patient may complain of a failure of accommodation, which shows itself by the need of stronger glasses, or of occasional attacks of blurred vision and halos about the light.

Symptoms of attack begin suddenly with a severe pain in the eye and head. There may be some temperature, nausea, and vomiting. The lids are swollen, the eyeball is deeply congested, cornea steamy and anæsthetic, anterior chamber very shallow, pupil dilated and oval in shape, and the iris discolored. The fundus can not usually be made out on account of the cloudiness of the media. Vision may be diminished in a few hours even to perception of light. Tension is very high. This attack lasts from a few hours to a few days; then all acute symptoms gradually subside, leaving the vision more or less permanently impaired. The other eye may be affected in a similar manner at any time.

Recurrence.—After a few weeks or months the acute attack is repeated, and if no treatment is given, the eye later will pass into a condition of subacute or chronic glaucoma.

Diagnosis.—Although acute glaucoma is not a common disease, the importance of differential diagnosis is evident from the fact that if the condition is mistaken for iritis and atropine prescribed, the results will be disastrous. The following table will assist the student. It applies to acute conditions.

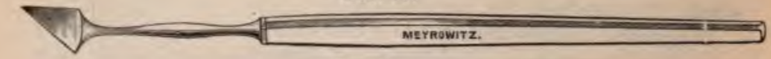
	Glaucoma.	Iritis.	Conjunctivitis.
Age	Over forty.	Any.	Any.
Tension	Plus.	Normal.	Normal.
Secretion	None, or watery.	None, or watery.	Mucopurulent.
Congestion . . .	General, especially scleral.	General, especially circumcorneal.	Conjunctival, especially of lids.
Cornea	Cloudy and steamy surface.	Cloudy.	Clear.
Anterior chamber	Shallow.	Unchanged.	Unchanged.
Iris	Discolored.	Discolored.	Unchanged.
Pupil	Dilated, oval.	Contracted, synechiæ.	Unchanged.
Pain	Severe, continuous.	Especially at night.	None.
Vision	Much reduced.	Somewhat reduced	Good.
Treatment . . .	Eserine, pilocarpine, iridectomy.	Atropine.	Astringent.

Prognosis.—After proper treatment, the results in acute glaucoma are usually satisfactory. It must be borne in mind, however, that the operation or after-treatment at times precipitates an attack in the other eye.

Treatment.—Persons over forty, complaining of rapidly failing accommodation, transient blurs, and halos should be kept under observation. In the acute attack the patient should be kept in bed and given a full dose of an opiate. Locally, eserine sulphate (0.5 per cent.) should be instilled at first every hour or two; later, three to five times a day.

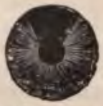
But if the tension remains high and vision becomes worse, **iridectomy** should be performed as follows: A general anæsthetic is necessary in acute glaucoma. After cleansing the eye, the speculum is adjusted. The fixation forceps are then placed in the conjunctiva below the cornea, and a lance-knife or bent keratome is introduced into the sclera above, about 1 mm. back of the clear cornea, and the point is passed into the anterior chamber over the iris. After withdrawing the knife, the iris forceps is introduced and the iris grasped near the pupillary edge and drawn out of the wound. The iris is then cut off with two snips of the scissors very near to the eyeball. The cut edges of the iris are replaced with a spatula, making the ideal keyhole iridectomy. Some operators use the Graefe cataract-knife for the incision.

FIG. 29.



Keratome.

FIG. 30.



Iridectomy for glaucoma. (De Wecker.)

CHRONIC INFLAMMATORY GLAUCOMA.

Definition and Symptoms.—After repeated attacks of acute glaucoma, or primarily, the tension may become permanently increased. There is more or less pain, enlargement of scleral vessels, shallow anterior chamber, oval, enlarged and immovable pupil. Vision is reduced or destroyed. Later, when there is no perception of light, very high tension, cataractous lens, wide pupil, and shallow anterior chamber, the condition is known as *Absolute Glaucoma*.

SIMPLE GLAUCOMA.

Synonyms.—Chronic noninflammatory glaucoma; Glaucoma simplex.

Etiology.—Age over forty. Hyperopic refraction, arteriosclerosis, and high arterial tension.

Pathology.—See Acute Inflammatory Glaucoma.

Subjective Symptoms.—The only complaint from the patient is gradual decrease in vision and halos about artificial light. There is no pain, only occasionally a sense of pressure.

Objective Symptoms.—The eye will be found free from congestion, except perhaps a few enlarged scleral vessels. The anterior chamber may or may not be shallow. The pupil may be normal and movable, or somewhat dilated. The lens and vitreous are clear. The optic nerve is atrophic, white or gray, and shows the characteristic condition known as "cupped disk." Just inside the scleral ring, which is broad, the nerve falls abruptly to a deeper level, and the bloodvessels will be seen dropping over this edge and appearing again at the bottom of the excavation. The arteries, if closely observed, will be seen to pulsate. The tension of the eye is increased, but not constantly, at times being quite normal. The field of vision is contracted concentrically, but more especially on the nasal side. There may be irregular contraction in any part of the field, and even isolated scotomata. The acuity of vision is usually reduced, although in some cases the field is much contracted before the vision is affected.

Course.—Both eyes are usually affected at about the same time. The disease continues over a number of years. If no treatment is undertaken, it ends in absolute glaucoma.

Diagnosis.—In some cases the appearance is very similar to atrophy of the optic nerve with excavation, and may only be distinguished by detecting increase of tension, which is not at all times present. In prescribing mydriatics such as atropine for a patient over fifty, the possibility of glaucomatous tendencies should be borne in mind, since these drugs tend to increase intraocular tension.

Prognosis.—Although the prognosis is poor, the disease will usually be arrested, or at least the rapidity of its progress decreased by operation. The success of operative treatment is in general proportionate to the size of the field of vision. On rare occasions the eye operated upon will become more rapidly worse. Sometimes there is no effect. Increased tension, with inflammatory signs, immediately after operation, is of rare occurrence. This latter condition is known as **malignant glaucoma,** and results in loss of the eye.

Treatment.—Eserine (0.2 per cent.) t. i. d., or pilocarpine (1 per cent.) t. i. d., should be given as temporary palliative treatment. An iridectomy, broad at the base of the iris, should be performed, as already described. It may be done under cocaine. Sclerotomy as a substitute for iridectomy may be tried: The eye is entered with a Graefe knife, as for cataract incision, but the knife is withdrawn before the flap is completed (anterior sclerotomy). Or, the knife is entered well back in the sclera and the eye pierced behind the lens (posterior sclerotomy). Of late, excision of the superior cervical ganglion (sympathectomy) has been performed for the relief of glaucoma with some success.

Secondary Glaucoma is generally due to mechanical causes, such as filling up of the iris angle, swelling of the lens, dislocation of the lens, hemorrhages, intraocular tumors, injuries, choroiditis, retinitis, and closure of the pupil.

Hemorrhagic Glaucoma.—A form which appears after retinal hemorrhages of various origin. Its treatment is unsatisfactory, iridectomy being generally disastrous from the occurrence of further hemorrhages.

QUESTIONS.

Into what varieties may glaucoma be divided?

Give cause, symptoms, and treatment of acute inflammatory glaucoma.

Give differential diagnosis between acute glaucoma, iritis, and conjunctivitis.

Give pathology, symptoms, diagnosis, prognosis, and treatment of simple glaucoma.

CHAPTER XVIII.

SYMPATHETIC OPHTHALMIA.

Definition.—A condition in which a healthy eye becomes the seat of a destructive inflammation transferred from the other eye, which has been the subject of a similar inflammation usually following a perforating injury of the eyeball. The injured eye is called the *exciting* eye; the other, the *sympathizing* eye. Although sympathetic ophthalmia is a comparatively rare disease, the possibility of its occurrence should not be overlooked on account of its terrible consequences.

Etiology.—Children are more susceptible than adults. The type of inflammation in the exciting eye is usually a chronic plastic iridocyclitis. This is produced in the majority of cases by a perforating wound involving the so-called "danger zone," or ciliary region—a zone ¼ inch wide about the cornea. There may or may not be retention of a foreign body within the eyeball. It may follow cataract extraction. The exciting eye may be one which has had a perforating ulcer of the cornea with incarceration of iris or ciliary body. Instances have been reported in which there was no evidence of perforation, traumatic or otherwise. Eyes destroyed by purulent inflammation, as a rule, do not produce sympathy.

Pathology.—The mode of transmission of the inflammation is still undecided. The theories are (*a*) irritation in the sympathizing eye, producing disturbances of nutrition and circulation, and, finally, inflammation through the agency of the intimate nerve relationship; (*b*) transferrence of germs by means of communication through the optic nerve or sheath; (*c*) transmission of infectious germs or toxins by means yet unknown. The last is the commonly accepted theory.

Symptoms of the Disease in the Exciting Eye.—The eye is more or less congested and painful. It is tender in the ciliary region when pressure is made through the upper lid; shows minus tension; posterior synechiæ or pupil blocked with exudate.

Symptoms of the Disease in the Sympathizing Eye.—There is a chronic inflammation involving the uveal tract (iris, ciliary body, and choroid). It may begin (*a*) with slight ciliary congestion, punctate deposits on Descemet's membrane, deep and cloudy anterior chamber, slightly dilated pupil, a few synechiæ and opacities in the vitreous (so-called serous iritis); (*b*) or the disease may begin at once as a plastic iridocyclitis with pain, ciliary tenderness, ciliary congestion, small and blocked pupil, opacities of vitreous, and later formation of bands and detachment of the retina and shrinking of the eyeball; (*c*) the disease may also appear first as a neuroretinitis.

Course.—Sympathetic ophthalmia appears between the third week and the sixth month after the original injury. The extreme limits are two weeks and twenty or more years. The second eye is likely to be attacked during a period of active inflammation in the exciting eye. Although there are usually some symptoms of sympathetic irritation (see page 127) before the genuine attack sets in, it often appears without warning.

The disease in the sympathizing eye runs a chronic course, with exacerbations.

Prognosis.—Sympathetic inflammation once established leads to blindness in the vast majority of cases. Those appearing as neuroretinitis are the most favorable.

Treatment.—The enucleation of a suspected eye, if we can be assured that the sympathetic process has not begun, is a positive preventive, and should be practised in cases of hopelessly extensive wounds. Further, an eye which has been the subject of an injury especially involving the ciliary body or containing a foreign body, and which shows signs of plastic iridocyclitis with ciliary tenderness and in-drawn scar, should be enucleated. Evisceration of the contents of the eyeball or section of the optic and ciliary nerves are advocated by some as a substitute for enucleation.

When sympathetic ophthalmia is established, enucleation of the exciting eye is usually considered of no value—at all
not be performed if any vision remains in the

exciting eye. After cessation of the disease the first eye may be the better. The patient should be treated with rest in bed, mercury—either by inunctions or internally—and by diaphoretics. The eye should be kept under the influence of atropine and hot fomentations. If the inflammation finally subsides without shrinking of the eyeball, an attempt may be made to obtain a clear pupil by iridectomy or iridotomy, but it will usually close again.

SYMPATHETIC IRRITATION.

Definition.—This is a distinct affection apart from sympathetic inflammation. It is a neurosis, showing in the sympathizing eye photophobia, lachrymation, symptoms of asthenopia, impaired accommodation, and contraction of the field of vision. In its simplest form it may be produced by irritation, such as arises from the presence of a foreign body on the cornea, or may appear when the exciting eye is one capable of producing sympathetic ophthalmia. These phenomena are likely to recur at intervals, and may be the precursors of the true inflammation. Hence blind offending eyes should be removed.

QUESTIONS.

Define sympathetic ophthalmia.
What is the mode of transmission from one eye to the other?
Describe the disease in the exciting eye and in the sympathizing eye.
What are the course and the prognosis?
Give the rules for enucleation of the offending eye.
What is sympathetic irritation?

CHAPTER XIX.

REFRACTION.

SUBJECTIVE EXAMINATION.

A **test-case** consists of lenses and prisms graded in strength.

Lenses.—A lens is made of glass or crystal, with at least one surface curved, and has the power of refracting or changing the direction of rays of light.

Prisms.—A prism is wedge-shaped, and bends rays of light toward its base. Prisms are numbered (*a*) according to the angle of the two sides; or (*b*) by the amount of angular deviation of a ray of light, which is produced by the prism: the latter may be considered about one-half the former; (*c*) deviation measured in one-hundredths of a given arc (centrad).

Spherical Convex Lens.—Usually biconvex. Converges rays of light to a focus. When the entering rays are parallel, the distance from the optical centre of the lens to the focus is called the principal focal distance. The stronger or more convex the lens, the shorter the focal distance.

Convex Cylindrical Lens.—May be represented by a section, cut in one plane, from a solid cylinder parallel to the axis of the cylinder. One surface would be plane, the other surface would have a convex meridian, and the other meridian at right angles to it—a straight line. The focus is determined for the meridian of greatest curvature, and is a line instead of a point.

Concave Spherical Lens.—Usually biconcave. Diverges rays of light and has no real focus. The focal distance is found by extending the divergent rays backward until they meet. The same principles then apply as for convex sphericals.

Concave Cylindrical Lens.—Corresponds to convex cylindrical lens, but has one concave meridian.

Numbering of Lenses.—Lenses are numbered according to two methods:

1. *The inch system,* which designates the strength by the inverse of the principal focal distance in inches: $\frac{1}{20}''$ signifies a lens whose focal distance is 20 inches.

2. *The diopter* (dioptry, dioptre) *system.* A lens with a focal distance of one metre is the unit. A lens of one-half this focal distance would be called 2 D.; one-quarter of this distance, 4 D.

One system may be converted into the other, approximately, by dividing the number of the lens, in either system, into 40—*e. g.*, 2 D. equals 20 inches, or, 10 inches equals 4 D. + signifies convex, and — concave, lenses.

Diagnosis with Lenses.—After recording the vision, as described on page 26, if it is $\frac{20}{20}$, one may conclude that the patient is either emmetropic or hypermetropic. If a convex spherical lens is held before the eye and the vision still remains perfect, the eye is hypermetropic and the strongest glass accepted measures the *manifest hypermetropia.* If no plus glass is accepted, the eye is probably practically emmetropic. If the vision is not equal to $\frac{20}{20}$, and the cause is a refractive error, this is either myopia, astigmatism, or high hypermetropia. Concave spherical lenses should be tried, and if the vision is made perfect, the weakest lens measures the *myopia.* If not made perfect, convex and concave cylinders should be tried at all axes until vision is improved. The improvement of vision by cylinders shows the presence of *astigmatism.*

OBJECTIVE EXAMINATION.

Ophthalmoscope.—The direct method is employed. The observer must learn to relax his accommodation. The accommodation of the patient usually relaxes under the examination, or a cycloplegic (atropine, 1 per cent., or homatropine, 2 per cent.) may be used. Hypermetropia is measured by noting the strongest convex lens of the ophthalmoscope through which the fundus may be clearly seen, and myopia by the weakest concave lens. Astigmatism gives a streaked appearance to the fundus. Each meridian may be measured, as is simple hypermetropia or myopia, by focussing upon the retinal vessels. With practice, the refraction may be estimated with great accuracy.

Skiascopy (Retinoscopy, Shadow Test).—A skiascope, or retinoscope, is a circular plane or concave mirror with a small central aperture. If the light is placed back of the patient, and the observer stands about 1.5 metres in front and reflects the light into the patient's eye, the pupil will show a red reflex. When the reflected light is moved slowly from side to side, the red pupil reflex will appear to move either in the same or in the opposite direction. If the reflex, or the shadow which borders it, moves in the same direction, when a plane mirror is used, the eye is hypermetropic. If in the opposite direction, the eye is myopic. The reverse is true if the concave mirror is used. By placing lenses before the patient's eye, one may determine the exact correction when there is no movement of the reflex. The different meridians of astigmatism may be separately measured in this way. Skiascopy is the most accurate method of determining refraction.

Ophthalmometer.—An instrument for the determination of variations in the curvature of the cornea by means of reflexes. As astigmatism is, for the most part, in the cornea, the instrument is of value in confirming other tests.

INDIVIDUAL ERRORS OF REFRACTION.

If parallel rays of light are brought to a focus on the retina, with the accommodation at rest, the refraction is normal and the eye is said to be *emmetropic*. If under these conditions the rays are not brought to a focus upon the retina, the eye is *ametropic*. The varieties of ametropia, or errors of refraction, are (I.) *hypermetropia*, (II.) *myopia*, and (III.) *astigmatism*. *Anisometropia* is a term commonly employed to denote that one eye differs markedly from the other in refraction.

I. HYPERMETROPIA.

Synonym.—Farsightedness.

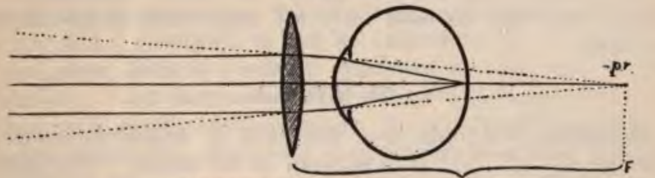
Definition.—A condition in which, with the accommodation at rest, parallel rays of light fall *behind* the retina.

Hypermetropia is a congenital defect, and never increases. The eyeball is too short in anteroposterior diameter for the refractive power of the media. Thus the focus falls behind the retina, but while the accommodation (see page 27) is strong, the focus may be brought forward.

Symptoms.—The patient complains of asthenopia, under which head are classed the symptoms produced by eye-strain: pain in the eyes, headache (especially frontal), blurring and running together of type, conjunctivitis, photophobia, tiring of the eyes, dizziness, etc.

Tests.—When the vision is tested at a distance, it will be found perfect unless the patient has lost his accommodation, but he will see as well with a convex glass. The strongest convex lens through which the patient sees as well as without, measures the **manifest hypermetropia.** A cycloplegic such as homatropine hydrobromate (2 per cent. solution) dropped into the eyes three times during an hour, or atropine sulphate (1 per cent.) three times a day for three days, will paralyze the accommodation, and the vision will then fall below the normal, because the patient can no longer use his accommodation and bring forward the focus upon the retina. The convex lens, which then gives him perfect vision, will be stronger than that for the manifest hypermetropia, and measures the exact refraction of the eye or **total hypermetropia.** The difference between the manifest and total hypermetropia is called the **latent hypermetropia.** As one grows older, the accommodation weakens, and the latent hypermetropia becomes less until about fifty or fifty-five, when the manifest equals the total, if it does not before.

FIG. 31.



Correction of hypermetropia.

Example.—If a patient under forty has perfect vision at distance—*e. g.*, $\frac{20}{20}$ with each eye, and sees as well with +2 D. spherical, and with no stronger lens, he is said to accept +2 D., or to have manifest hypermetropia of that amount. If the eyes are put under the influence of atropine, his vision will be reduced to perhaps $\frac{20}{70}$, but with +3.5 D. is brought to $\frac{20}{20}$. He thus has total hypermetropia +3.5 D. and latent +1.5 D.

Treatment.—In young persons it is well to examine the eyes with a *cycloplegic.* When the observer is skilled in the use of the ophthalmoscope, skiascope, and ophthalmometer, the use of cycloplegics becomes less necessary, especially in adults. In some cases, particularly those with muscular errors, it is best to give the correction for total hypermetropia (full correction). But since the distant vision will be blurred with these lenses when the effects of the atropine have passed, it is generally customary to correct the manifest error and a little of the latent, in addition.

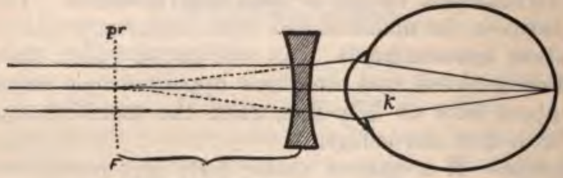
In children the glasses should be worn constantly. *In adults* they may be used only for close work if the error is not large.

II. MYOPIA.

Definition.—Myopia is a condition in which the focus for parallel rays of light falls in front of the retina. The antero-posterior diameter is too long. It is a diseased condition.

Etiology.—Myopia may be congenital, but is generally acquired, beginning usually between the ages of eight and fifteen. In the latter form there may be hereditary predisposition, but improper use of the eyes is the direct cause.

FIG. 32.



Correction of myopia.

Complications.—There is bulging of the posterior segment of the eyeball (posterior staphyloma). In high degrees this is accompanied by a chronic inflammatory process (choroiditis), which produces changes in the fundus, especially at the macula and about the nerve. There is also a tendency toward hemorrhages in the fundus, detachment of the retina, and opacities of the vitreous. Insufficiency of the internal recti is a common complication, and at times divergent strabismus occurs.

Symptoms.—The young patient begins to complain, in school, of poor vision at a distance. There is usually no asthenopia. If the disease has reached a high degree, near work is held very close to the eyes.

Tests.—The distant vision is found considerably reduced, while perfect at the near point. Concave spherical lenses bring the distant vision to normal if there are no complications. The weakest concave lens which gives perfect vision measures the myopia. Atropine is not so important as in hypermetropia, since there is a natural tendency to relax the accommodation. Low degrees of myopia, however, should be tested with a cycloplegic, because *spasm of accommodation*, or false myopia, is possible, and gives poor vision, improved by weak concave lenses.

Course.—Myopia may increase through youth and reach a high degree before the twenty-fifth year. It is then called progressive. When rapidly progressive, it is known as malignant.

Treatment.—Near work should be restricted, and the eyes used only under the most favorable conditions as to proper position at desk, illumination, etc. Attention should be given to the general health and exercise out-of-doors prescribed. For the *lower degrees* of myopia, it is commonly agreed that the full correction of the error should be worn constantly. In *high degrees* and in patients over forty, a weaker glass should be given for near work. The crystalline lens may be removed by absorption or extraction in myopia over —15 D. This more or less exactly neutralizes the myopia.

III. ASTIGMATISM.

Definition.—A condition in which one meridian varies from another in refractive power. Astigmatism is largely in the anterior surface of the cornea. The corneal surface may be likened to that of the bowl of a spoon. Some astigmatism may be present in the lens. There may be irregularities in the surface of the cornea (irregular astigmatism); but, in the ordinary acceptance of the term (regular astigmatism), the extremes of curvature are at right-angles to each other. The axis of the astigmatism, as of the cylinder which corrects it, is that meridian which is nearest the emmetropic. There is no one point as a focus for the astigmatic eye, but there are two foci—one behind the other, and each a line at right-angles to the other. Astigmatism changes only gradually through life.

Varieties.—*Simple hyperopic astigmatism:* one meridian emmetropic; the other, at right-angles, hypermetropic.

Simple myopic astigmatism: one meridian emmetropic; the other myopic.

Compound hypermetropic astigmatism: one meridian hypermetropic; the other more hypermetropic.

Compound myopic astigmatism: one meridian myopic; the other more myopic.

Mixed astigmatism: one meridian hypermetropic; the other myopic.

Symptoms.—There is complaint of poor vision, of seeing lines in one direction better than another, and of asthenopia.

Tests.—The vision is affected according to the degree of the astigmatism, both for near and distance. Cylinders at the proper axis will give improvement in vision. Atropine should be used for the examination under the same conditions as in hypermetropia. A further subjective test is made with a card having lines which radiate in all directions from a common centre, like spokes of a wheel. The astigmatic eye will not see all the lines with equal distinctness, and cylinders can be tried at all axes until the lines appear alike.

Treatment.—It is desirable to order as weak a cylinder as possible.

ERRORS OF ACCOMMODATION.

Presbyopia.—The near point of monocular vision (punctum proximum) is about two and one-half inches in the young child. This near point gradually recedes until between forty and forty-five it is no nearer than fifteen inches, which is the average reading distance. When the near point of accommodation recedes beyond this point and vision is not clear within this limit, the condition becomes unpleasant for reading, writing, and other near work, and is known as *presbyopia*. If a patient at forty-five can accommodate to a point 14 inches from the eyes, he has a range of accommodation from infinity to a distance of 14 inches, or 2.75 D. If at fifty his near point is 30 inches, his range is only 1.25 D., and he needs a lens to give him the required ranges—*i. e.*, +1.50 D., which would make +2.75 D., or bring the near point to 14 inches. When he has no accommodation at fifty-five, he will need +2.75 D. for near work.

QUESTIONS.

What is a lens? A prism?
Describe four varieties of lenses.
How are lenses numbered?
Give rule for changing one system into the other.
Describe the use of the ophthalmoscope, retinoscope, and ophthalmometer.
Give method of subjective examination.
Define hypermetropia.
Explain the accommodation.
What are manifest, total, and latent hypermetropia?
Give tests for hypermetropia. What is the treatment.
Give the definition, etiology, complications, and treatment of mypopia.
What is spasm of accommodation?
Define astigmatism. State varieties and tests.
Describe presbyopia.

CHAPTER XX.

THE MUSCLES OF THE EYE.

THE physiologic action of the external muscles of the eyeball is as follows:

Internal rectus turns the eye inward (adduction).

External rectus turns the eye outward (abduction).

Superior rectus turn the eye upward and inward (adduction), and rotates the upper extremity of the vertical meridian inward (wheel rotation or torsion).

Inferior rectus turns the eye downward and inward, and rotates the upper end of the vertical meridian outward.

Superior oblique rotates the upper end of the vertical meridian inward, and turns the eye downward and outward.

Inferior oblique rotates the upper end of the vertical meridian outward, and turns the eye upward and outward.

The monocular *field of fixation* is the area outlined by the limits of movement of each eye when the head is at rest.

Errors of motility comprise: I., Insufficiency; II., Strabismus; and III., Paralysis.

I. INSUFFICIENCY.

Definition.—A condition in which one or more muscles are lacking in power, but in which binocular vision is maintained in spite of the error. There is a tendency for the eyes to deviate, but this is controlled by constant effort.

Etiology.—Temporary depression in the general health, overuse of the eyes, insufficient innervation or mechanical disadvantage, such as malposition of muscular attachment.

Varieties.—The following terms are used to describe the varieties of insufficiency:

Orthophoria is the condition of normal balance.

Heterophoria, imperfect balance or insufficiency.

Exophoria, tendency for eyes to spread. Insufficiency of the internal recti.

Esophoria, tendency to converge. Insufficiency of the external recti.

Hyperphoria, tendency for eyes to deviate vertically. Insufficiency of superior or inferior recti.

Symptoms.—Pains in the eyes, headaches, running together of print, diplopia, dizziness; in general symptoms classed under the head of asthenopia.

Tests.—(*a*) Some patients with heterophoria will *spontaneously* or voluntarily allow the eyes to separate or many will develop diplopia when a red glass is held over one eye. (*b*) *Cover-test* (see p. 31). (*c*) *Vertical diplopia* or Graefe test consists in holding a prism of 8° or 10° with base down over one eye. This throws the image of that eye upward over the image of the other. If the lateral muscles are in equilibrium the upper image will lie vertically over the other. The amount of esophoria or exophoria may be measured by a prism with base out or in which brings the upper image over the under one. (*d*) *Adduction and Abduction Tests.*—If a prism is held horizontally over either eye with the base either in or out and eyes fixed on object twenty feet distant, horizontal diplopia will be produced; but if the prism is not too strong, the two images will run together and fuse into one. The strongest prism with the base *in* through which the two images may be made to fuse measures the fusion power of the external recti (abduction). This should be about 7° or 8°. The strongest prism with base *out*, through which the images fuse, measures the internal recti (adduction). This should be 20° or 30°. The latter test is often deceptive. The same method may be used to test the elevators and depressors by holding the base of the prism either up or down. Fusion power should be 3° or 4°. A phorometer is an instrument constructed to hold suitably prisms used for the tests just described.

Treatment.—Attention should be given to the general health. Errors of refraction should be carefully corrected. If there is no improvement, so-called gymnastic exercises may be prescribed. The patient practises by fusing the images which are produced by the strongest prism, making fusion possible, with the base over the muscle to be strengthened. The results are sometimes gratifying, but not likely to be permanent. The constant use of a prism as an eyeglass may be of value in some cases. A prism of about one-half the error, if it is not too great, is generally suitable. If the case is not improved by the treatment above outlined, faithfully carried out operative measures are to considered. If the external recti show abduction over 12° with weak adduction, the external rectus of one eye may be severed by a complete tenotomy.

If the external recti show abduction as low as 1° or 2° with adduction strong, the internal rectus may be cut with caution. If excessive effect is produced at the time of the operation, it may be limited by a suture. So-called partial tenotomies consist in cutting a portion of the tendon. They yield no permanent results.

II. STRABISMUS.

Synonym.—Squint.

Definition.—A condition in which both lines of sight are not directed toward the same object of fixation.

Varieties.—The common varieties are *convergent strabismus* (internal squint), where one eye is turned inward toward the nose; *divergent strabismus* (external squint), one eye turns outward toward the temple. *Deorsumvergent strabismus* (downward) and *sursumvergent strabismus* (upward) are uncommon. A strabismus is said to be *concomitant* when the angle of deviation between the two lines of sight remains the same for any point in the field of fixation equally distant from the eyes. *Periodic*, when the squint is not constant. *Alternate*, when either one or the other may be the squinting eye.

CONVERGENT STRABISMUS.

Etiology.—Generally appears between the ages of two and five. At first periodic, later constant. Hypermetropia is generally the refractive condition of the eyes. The squinting eye is often amblyopic (having poor vision). This is regarded by some as the result of nonuse (amblyopia ex anopsia), and therefore secondary. By others it is considered a congenital defect, and partly the cause of the strabismus.

Symptoms.—The child does not complain of diplopia, except rarely at the beginning, because the image of the squinting eye is disregarded.

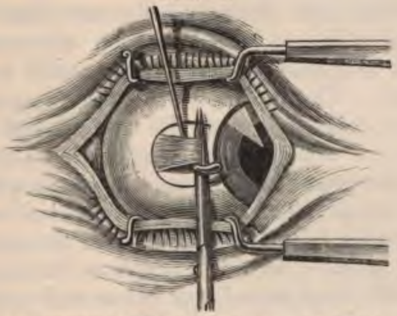
Tests.—Measurement of the degree of squint may be made by noting the distance on the lower lid at which the centre of the cornea lies from the place which corresponds to that of the other eye. The angular deviation may also be measured by means of the reflex from the cornea by passing *ndle along the* arc of a perimeter.

Treatment.—Nonoperative.—Convergent strabismus may be treated in very young children when it first appears by placing a cover over the better eye and forcing the child to use the squinting eye. It is important, when the child is old enough, that glasses should be worn to correct fully the errors of refraction. There is usually hyperopia, or hyperopic astigmatism. Under such treatment a number of cases will recover.

Operative treatment consists in performing tenotomy of the internal rectus of one or both eyes, and sometimes an advancement of the external rectus.

Tenotomy.—The conjunctiva over the insertion of the tendon is seized with a toothed forceps about 7 mm. from the edge of the cornea. A small opening is made with the scissors, exposing Ténon's capsule. This is opened in a similar manner, and the tendon may then be caught up by passing a strabismus hook beneath it. The tendon should be completely cut close to the eyeball. A suture may be used to bring together the edges of the conjunctival wound. The opening in the conjunctiva and capsule is sometimes made at the edge of the tendon and the muscle cut by passing the scissors underneath the conjunctiva (subconjunctival tenotomy). The effect of a tenotomy upon a squint is not always easy to predict with certainty. Remembering, however, that the edge of the cor-

FIG. 33.



Tenotomy of an ocular muscle. (Veasey.)

nea should normally turn on either side to the inner and outer canthus, any decrease or increase of motility can be estimated. Such indications are the best guides for operation. If tenotomy of the internal rectus of the squinting eye does not produce sufficient effect, the other eye may be operated upon after waiting at least two months. At times, when the motility of the squinting eye is markedly deficient outward, advancement (see p. 137) of the internal rectus is indicated. As there is a tendency toward divergence in later years, it is safer to leave a little convergence.

DIVERGENT STRABISMUS.

Etiology.—May appear at any age. Often associated with myopia. Found after loss of vision in an eye from injury or disease and after faulty operation for convergent strabismus.

Treatment.—In the majority of cases tenotomy of the external rectus will not suffice, but an advancement of the internal rectus is necessary in connection with it.

Advancement.—The tendon of the opposing muscle is first cut. An incision is made over the tendon, and sometimes an elliptical piece of conjunctiva is excised. Ténon's capsule, thus exposed, is opened and the muscle raised upon two hooks. One suture is passed through the conjunctiva on the side of the wound farthest from the cornea, then through the middle of the muscle about $\frac{1}{8}$ inch or more from its insertion. Two other sutures are then passed, one below and one above. The tendon is severed from its attachment to the eyeball and a piece cut off nearly down to the sutures. The first suture is then passed in a horizontal line under the conjunctiva on the corneal side, taking a firm hold on the episcleral tissue and coming out near the cornea. The other two are passed in the same way above and below. The sutures, when tied, will advance the muscle to produce the effect desired. Sutures should be left in place about seven days.

III. PARALYSIS OF THE OCULAR MUSCLES.

Varieties.—Classification may be (*a*) according to the nerve affected. The third nerve supplies all the muscles (including the sphincter pupillæ, ciliary muscle, and levator palpebræ) except the external rectus and superior oblique, which are supplied by the sixth and fourth respectively; (*b*) according to the muscle paralyzed.

Internal ophthalmoplegia is a term used to designate paralysis of the intrinsic muscles, the sphincter, and ciliary muscle; and **external ophthalmoplegia,** paralysis of the extrinsic muscles; **total ophthalmoplegia,** when both *intrinsic* and *extrinsic* muscles are paralyzed.

Etiology.—Paralysis may be *congenital,* especially of the external rectus and levator palpebræ (ptosis). In the *acquired* form, syphilis is the most common cause. Other causes are traumatism, locomotor ataxia, tumors, hemorrhages, and toxic conditions.

Pathology.—The paralysis may be cortical, nuclear, in the nerve-trunk, or peripheral.

Subjective Symptoms.—The patient suffers most from diplopia, dizziness, and confusion of vision.

Objective Symptoms.—The excursion of the affected eye is limited. The head is held habitually away from the side of the paralyzed muscle in order to avoid diplopia. The character of the diplopia may be tested as follows: A red glass is placed over the paralyzed eye. The red image is called the false image. The false image appears to the patient to occupy a position, relative to the true image, which represents the physiologic action of the muscle paralyzed. For example: if the right external rectus is paralyzed, there will be diplopia to the right of the median line, and the image of that eye will be to the right and on a level with the other image, which represents the action of the right external rectus—*i. e.,* turning the eye to the right horizontally. The above rule will be found applicable to all cases except where secondary contractions have taken place.

Treatment.—In syphilitic cases mercury and iodide of potassium should be given in full doses. In other cases sodium salicylate, mercury, iodide of potassium, and strychnine. Sometimes, in mild cases, wearing of a prism will help to fuse the images. At times a cover must be worn over the paralyzed eye. Operations are usually unsatisfactory.

NYSTAGMUS.

Definition.—An involuntary oscillatory movement of the eyeball, usually from side to side (lateral nystagmus), occasionally up and down (vertical), or with wheel rotation (rotary).

Etiology.—The condition is usually *congenital* and associated with poor vision. It is sometimes *acquired* when the eyes are used under poor illumination (miners' nystagmus).

Treatment.—There is no treatment for the former. For the latter, change of occupation.

QUESTIONS.

What is the physiologic action of each of the ocular muscles?

Define insufficiency, and give terms used to describe varieties.

Name four tests for heterophoria.

Give the treatment for heterophoria.

What are the common varieties of strabismus?

Give the cause, etiology, symptoms, and treatment of convergent strabismus.

Describe operations for tenotomy and advancement.

How may paralysis be classified?

Give rule for determining the muscle affected.

What is nystagmus?

THE EAR AND ITS DISEASES.

CHAPTER I.

ANATOMY AND PHYSIOLOGY.

For the **convenience of study** the organ of hearing may be considered as being composed of (1) a **conducting apparatus**—consisting of the auricle, external auditory meatus, membrana tympani, ossicles, and the tympanum, together with the connecting cavities of the Eustachian tube and the mastoid process—by means of which certain forms of ethereal vibrations are collected and transmitted inward to (2) a **perceptive mechanism**—divided into the vestibule, semicircular canals, and cochlea, with its filamentous terminations of the auditory nerve—through the agency of which the vibrations are recognized and the resulting stimulation is conveyed to the auditory centre of the brain and there interpreted as sound.

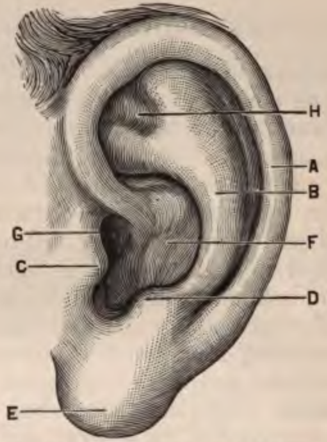
A further **clinical subdivision** considers the conducting portion as being composed of an **external ear** (auricle and external auditory meatus), a **middle ear** (membrana tympani, ossicles, and the tympanic cavity, with its connecting Eustachian tube and mastoid process), while the perceptive mechanism constitutes the **internal ear** or **labyrinth.**

THE CONDUCTING APPARATUS.

The External Ear.

The Auricle.—To secure the best concentration and conduction of the sound-waves, the external ear is an oval, funnel-shaped organ with the convexity of the auricle pointing inward toward the middle ear or tympanum. In structure, with the exception of the lobule (Fig. 33), which is composed of a network of connective tissue interposed with globules of fat, the **auricle** or **pinna** consists of a convoluted, cartilaginous framework, attached to the temporal bone by ligaments and rudimentary muscles, and covered by a thin, firmly adherent reflection of the integument from the side of the head, preserving the ridges and depressions of the substructure, which are named as indicated in the accompanying illustration.

FIG. 33.



Auricle: A, helix; B, antihelix; C, tragus; D, antitragus; E, lobule; F, concha; G, orifice of the external meatus. (Politzer.)

The **external auditory meatus** is a canal, which leads from the concha inward about one inch and a quarter, terminates with a membrane which separates the external ear from the tympanic cavity (Fig. 34). The outer third of this passage, which is a prolongation inward of the auricle, is cartilaginous, and forms an angle with the inner portion, which is bony in the adult. *Hence, the auricle must be pulled* *upward, backward, and outward to make the meatus straight, when examining the interior of the canal or the terminating membrane.* The inner two-thirds of the meatus is membranous in the infant, with the exception of the distal end, which is formed by a bony ring, the **annulus tympanicus**, which with development gradually elongates outward, replacing the membranous passage and forming the osseous portion of the adult ear, which is about three-quarters of an inch in length. A prolongation inward of the skin of the auricle forms the lining membrane of the meatus, which is thin and firmly attached in the bony portion. In the cartilaginous part numerous hairs, together with sebaceous and ceruminal glands, are present, which normally prevent to a marked extent the entrance or retention of foreign bodies within the canal.

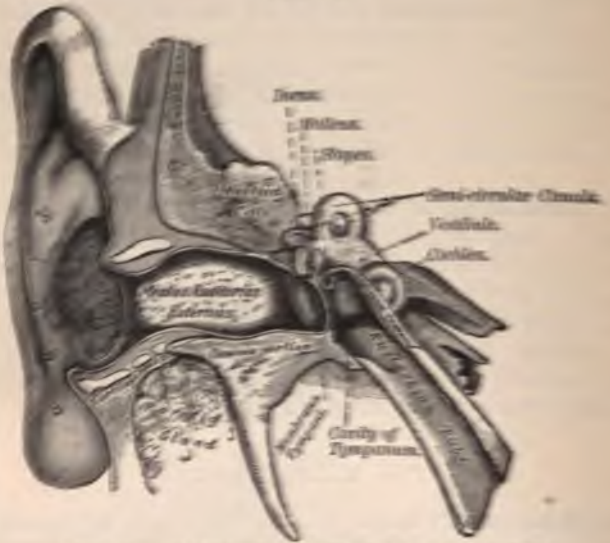
The **blood-supply of the auricle** and the outer end of the meatus is derived from the superficial temporal and the posterior auricular arteries, while the bony portion of the canal is supplied with branches from the internal maxillary.

The **nerve distribution of the auricle** is from the deep posterior auricular and auricularis magnus, while that of the external auditory meatus is derived mainly from the auriculotemporal branch of the inferior maxillary nerve.

THE MIDDLE EAR.

The Membrana Tympani.—Situated at the inner end of the external auditory meatus, and separating it from the tympanic cavity, is a pearly gray, oval-shaped membrane, concave on its outer surface, and placed obliquely to the axis of the canal so that the upper and posterior portion is the least removed from the examiner (Fig. 34). Its **structure** consists of an outer dermal surface continuous with that of the meatus, a middle fibrous membrane, and an inner mucous covering, which is reflected from the lining membrane of the tympanum. Within these layers rests the **long handle of the malleus**, the outlines of which are faintly followed from above, downward, and backward to its termination at the centre of the memrana tympani is a slight depression, **umbo**, from which oint a triangular, glistening area projects downward and forard to the periphery of the membrane (Fig. 35). This **cone light**, as it is termed, is a reflection of the rays from the rface of the membrana tympani, due to its smoothness, liquity of position, and contour of surface. At the upper

Fig. 34.

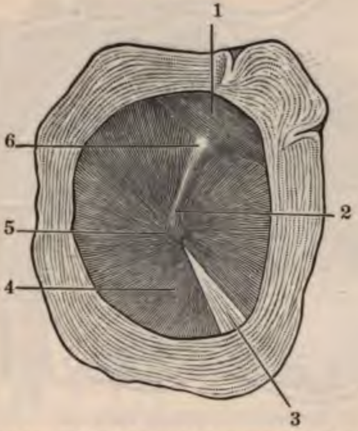


A front view of the organ of hearing (right side). (Gray.)

d of the handle is observed a yellowish-white projection, ort **handle of the malleus**, from which point thin folds of embrane pass antra-supra-posteriorly to the upper wall of e canal, constituting the **membrana flaccida** or **Shrapnell's embrane**. The membrana tympani not only serves as a otection to the delicate structures within the tympanum, t also receives the sound-vibrations from without and transits them to the ossicular chain of the middle ear.

The Tympanum.—Just beyond the membrana tympani, which forms the greater part of its outer wall, is situated, within the petrous portion of the temporal bone, **the tympanum,** an irregular cavity having its greatest diameters antra-posteriorly and supra-inferiorly, with an inward depth of about one-eighth of an inch to the promontory from the drum membrane (Fig. 36). The upper portion of this cavity, the **attic,** lies immediately below the middle lobe of the brain, separated from it by a thin layer of bone, which forms the roof of the tympanum. The lower portion of the tympanic cavity is termed the **atrium.** The floor, narrower than the roof and pierced by the Jacobson nerve, rests just above the jugular fossa. The outer wall, formed largely by the membrana tympani and the annulus tympanicus, into which bony ring the membrane is inserted, contains the Glaserian fissure, the iter chordæ posterius, and the iter chordæ anterius, the latter two being the openings through which the chorda tympani nerve enters and emerges from the tympanic

FIG. 35.

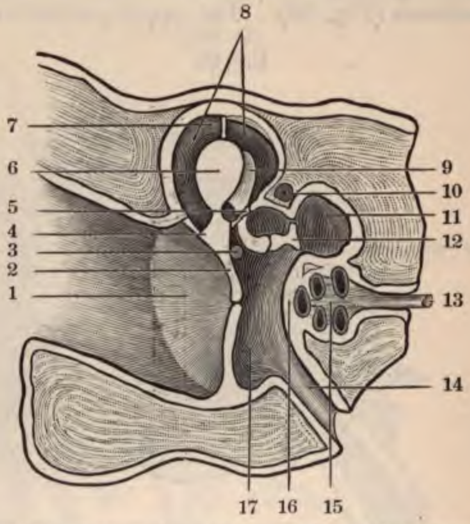


The normal membrana tympani (right ear): 1, membrana flaccida; 2, long handle; 3, cone of light; 4, membrana vibrans; 5, umbo; 6, short handle.

cavity in its course across the inner surface of the drum membrane between the long handles of the malleus and incus (Fig. 36) on its way to the submaxillary gland.

Separating the tympanum from the internal ear, the inner wall presents the following points of interest, as illustrated in Fig. 37:

FIG. 36.



The middle and internal ear: 1, membrana tympani; 2, long process of malleus; 3, chorda tympani nerve; 4, short process; 5, tendon of tensor tympani muscle; 6, malleus; 7, ligament; 8, attic; 9, incus; 10, facial nerve; 11, vestibule; 12, stapes; 13, auditory nerve; 14, Eustachian tube; 15, cochlea; 16, promontory; 17 atrium (right ear, anterior view).

(1) The **fenestra ovalis,** which opens into the vestibule of the internal ear, but is occupied, in the recent state, by the foot-plate of the stapes (Fig. 37).

(2) The **fenestra rotundum,** closed by a thin membrane (membrana tympani secondaria), beyond which lies the cavity of the scala tympani of the cochlea.

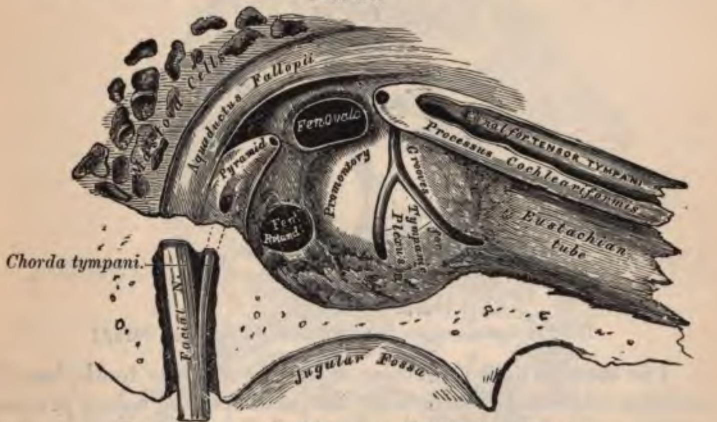
(3) The **promontory,** a convexity of the inner wall, formed

by the first turn of the cochlear spiral, which is situated just beyond the inner wall (Fig. 36).

(4) The **Aquæductus Fallopii,** a small canal through which the facial nerve passes, marked by a bony ridge along the supraposterior part of the inner wall.

(5) The **Pyramid,** a bony, cone-shaped projection, which contains the stapedius muscle.

FIG. 37.



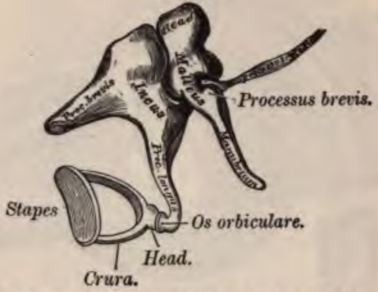
View of inner wall of tympanum (right ear). (Gray.)

The **anterior wall,** which is interposed between the carotid canal and the tympanum, presents the **tympanic orifice of the Eustachian tube, canal for the tensor tympani,** and the **processes cochleariformis,** which is a thin, bony partition separating the two canals, while the **posterior wall,** which has the greatest height, reveals in its upper portion a passage, **antrum,** through which the vault of the tympanum, **attic,** communicates with the **cells of the mastoid process,** situated posteriorly.

The Ossicles.—Connecting the membrana tympani with the fenestra ovalis of the inner wall, an ossicular chain, consisting of the **malleus, incus,** and **stapes,** serves to transmit the vibrations imparted to the drum membrane across the tympanic cavity to the labyrinth or internal ear. Fig. 38 illustrates very well the general shape, relative size, and position of the bones.

The **malleus** consists of (1) a *head*, which articulates with the incus; (2) a *neck*, constricted portion below the head; (3) a *long process, handle or manubrium*, which is embedded in the layers of the membrana tympani; (4) a *short process, processus brevis*, which presents at the upper end of the manubrium; and (5) a *processus gracilis*, which lies in the Glaserian fissure.

FIG. 38.



The small bones of the ear, seen from the outside. (Gray.)

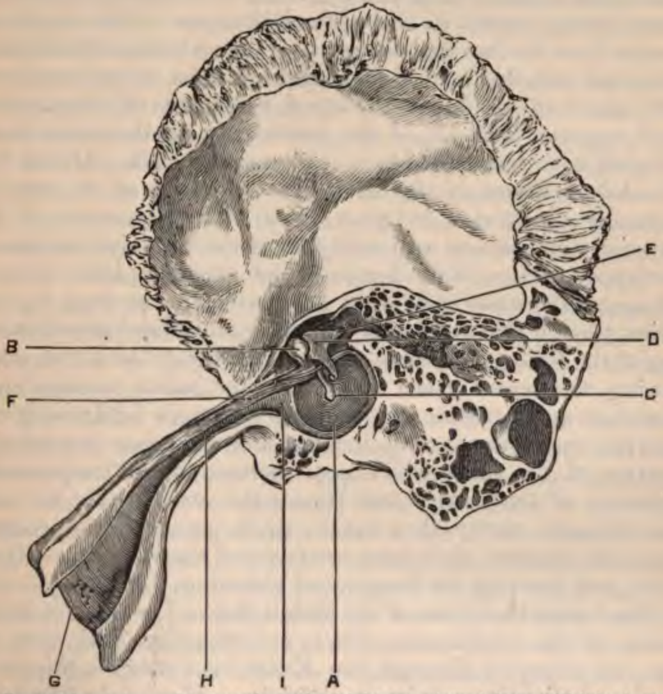
The **incus**, which resembles in outline a molar tooth, has (1) a *body*, which articulates with the malleus; (2) a *long process*, which articulates with the head of the stapes; (3) a *short process*, which has a ligamentous attachment at the lower edge of the antrum on the posterior wall of the tympanum.

The **stapes**, as its name indicates, is stirrup-shaped, and presents (1) a *head*, which articulates, as shown above; (2) a *foot-plate*, which occupies, in the recent state, the fenestra ovalis, being attached to the surrounding bony wall by an intervening annular ligament; and (3) the *crura*, which connect the head and the foot-plate.

The Joints and Ligaments of the Ossicles.—The ossicles are held in articulation and suspension within the tympanic cavity by means of ligaments, which, however, permit freedom of ossicular vibration in the normal state; but in certain diseased conditions the ligaments about the articulations become thickened, so that the joints assume a stiffened or even ankylosed condition, which interferes markedly with the ease of mobility, thus lowering the function of hearing even to inaudition if the process be sufficiently extensive.

The Eustachian Tube.—With an opening in the anterior wall

FIG. 39.



Eustachian tube and tympanic cavity (right ear, viewed from within): A, membrana tympani; B, head of the malleus; C, lower end of the handle of the malleus; D, body of the incus; E, short process of the incus; F, tensor tympani muscle; G, pharyngeal opening of the tube; H, isthmus of the tube; I, tympanic opening of the tube. (Politzer.)

of the tympanum (Fig. 39), an osseous canal passes, from this point, inward, forward, and downward through the petrous bone, when it merges into a cartilaginous canal, which terminates in a funnel-shaped protuberance, with a slit-like orifice, located in the nasopharynx, just back of the inferior turbinated body (Fig. 53). At the junction (**isthmus**) of the **cartilaginous** and **osseous portions**, the canal has a diameter, in the recent state, of about one-twelfth of an inch, which gradually increases toward the tympanic and pharyngeal ends, where it measures one-eighth to one-quarter of an inch, the slit-shaped orifice of the latter being greatest in the vertical direction. This condition results from the fact that the cartilaginous portion, which forms the inner two-thirds of the Eustachian tube, is not composed throughout of cartilage; the lower two-thirds of the anterior wall, together with all of the inferior, is membranous, intermingled with muscular fibres of the tensor palati. Hence, the slit-shaped lumen of the cartilaginous portion of the tube is normally obliterated in most cases through contact of the anterior membranous wall with that of the posterior, excepting the upper portion of the lumen, which remains patent because of its cartilaginous wall, thus preserving the passage at this point, though it may be obliterated by an inflammatory swelling of the lining mucous membrane. During the act of swallowing, the **tensor palati** and the **levator palati** muscles open the pharyngeal portion of the Eustachian tube by drawing the anterior membranous wall forward and elevating the inferior portion of the tube, which acts increase the antra-posterior diameter of the passage and permit the entering air to reach the tympanic cavity, thus equalizing the atmospheric pressure upon the internal and outer surfaces of the membrana tympani, and favoring its freedom of vibration.

The Lining Membrane of the Middle Ear.—The mucous membrane of the tympanum, which is continuous with that of the nasopharynx through the Eustachian tube, is thin, ciliated, and slightly vascular in character. It not only forms the lining investment of the tympanic cavity, but also extends to the spaces of the mastoid process through the intervening antrum, and forms a covering of the ossicles, intratympanic muscles, and ligaments. The resulting reduplications or folds of the mucous membrane are of special interest in inflammatory conditions of the tympanum, and *especially those in the attic*, which are large and numerous, as they form very favorable conditions for the development and retention of an infective process, as will be shown later. In the tympanic portion of the Eustachian tube the lining membrane is also thin, smooth, ciliated, and firmly adherent, while in the cartilaginous extremity it assumes the thick, vascular, and loosely attached character of the nasopharyngeal mucous membrane, through which route inflammatory conditions of the latter frequently extend to the tympanic cavity.

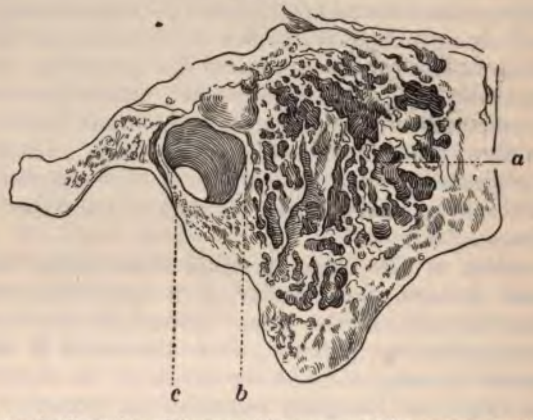
The Intratympanic Muscles.—Only two muscles, the tensor tympani and the stapedius, are present within the ear. The **tensor tympani** (Fig. 36) obtains its origin from the upper wall of the Eustachian tube and the sides of the canal through which it passes; emerges from the orifice of its canal just above the Eustachian opening; curves around the projecting processus cochleariformis at nearly a right angle; crosses the tympanic cavity; and makes its attachment to the inner border of the long handle of the malleus just below the neck. The **stapedius muscle** originates within the bony canal of the pyramid (Fig. 37), and forms an insertion into the neck of the stapes through an intervening tendon.

The **action of the tensor tympani** alone draws the drum membrane inward, thus rendering it more taut, and also forces the foot-plate of the stapes inward, thereby increasing the intralabyrinthine pressure, while the **action of the stapedius muscle** is antagonistic to the action of the former, as it tends to draw the foot-plate outward, the posterior margin serving as a fulcrum, thus lessening the labyrinthine tension. It is therefore probable that the function of these muscles is to preserve an uniformity of pressure within the labyrinth under the varying conditions of atmospheric force and intensity of sound.

The Arteries.—The blood-supply of the tympanum is derived mainly from (1) the **tympanic branch of the internal maxillary,** which enters the cavity through the Glaserian fissure, supplying the anterior wall and the tympanic end of the Eustachian tube, and anastomosing at the periphery of the membrana tympani with the stylomastoid branch of the posterior auricular; (2) the **superficial petrosal from the middle**

meningeal, which reaches the attic through the petrosquamous suture, and is distributed to the roof of the tympanum, the malleus and incus, and the inner wall through which, according to Politzer, "vascular connections are kept up between the middle ear and the labyrinth"; (3) the **tympanic branch of the internal carotid**, which anastomoses with the tympanic and Vidian branches of the internal maxillary upon the floor and inner wall of the tympanum. In addition to the supply indicated above, the Eustachian tube receives the pharyngeal branch of the external carotid, a few small branches from the internal carotid, and the descending palatine and pterygopalatine branches of the internal maxillary artery.

FIG. 40.



Vertical (sagittal) section of the mastoid process and the osseous meatus: *a*, mastoid cells; *b*, posterior wall of the osseous meatus; *c*, anterior wall of the osseous meatus. (Politzer.)

The Nerves.—Through the otic ganglion the trifacial supplies the tensor tympani, a filament from the facial innervates the stapedius muscle, while the lining membrane of the tympanum and the Eustachian tube receives its supply from the tympanic plexus.

***The Mastoid* Process.**—Immediately behind the auricle and somewhat inferiorly is located a large, rounded, bony protuberance, at the lower tip of which the sternocleidomastoid muscle forms an attachment. Its inward structure is usually composed of a varying number of pneumatic spaces or cells, irregular in size and shape (Fig. 40), which freely communicate with each other through openings in their intervening walls and also with the tympanic cavity through the antrum. Some mastoids are formed almost entirely of air-cavities, while others contain but few or no spaces, which is usually true of the infantile process, but with development the chambers are gradually formed and enlarged. *In the child* the inner wall of the mastoid, which separates the air-cavities from the lateral or sigmoid sinus of the brain, is quite thick, while the opposite outer wall is thin and often imperfect. Hence, inflammatory conditions within the mastoid rarely extend inward to the meninges in the youthful patient, but tend to pass outward through the opposite wall, a circumstance which is indeed fortunate. The anatomical conditions are reversed in the adult, though the firmer and harder structures form a strong barrier to the passage of infection inward. Since the mucous membrane lining the pneumatic spaces of the mastoid is continuous with that of the tympanic cavity through the antrum, infective processes of the tympanum frequently extend backward and involve the chambers of the mastoid process.

The **lateral sinus**, which courses along the inner wall of the mastoid, the **meninges of the brain**, which are separated from the antrum by only a thin lamina of bone, and the **facial nerve** in its passage through the aquæductus Fallopii, are structures of special importance to be considered in inflammatory conditions and surgical procedures.

THE PERCEPTIVE MECHANISM.

The Internal Ear.

Just beyond the inner wall of the tympanum a group of osseous cavities and curving canals receive the peripheral end-organs of the auditory nerve through which the sound-vibrations imparted from without are recognized and conveyed to the brain. The **labyrinth**, so termed from its complexity of shape, consists of an **osseous portion** (vestibule, semicircular canals, and cochlea) within which a **membranous portion** (utricle, saccule, semicircular passages, and scala media), filled with a fluid, forms a loose inner lining, being separated from the bony walls by an intervening perilymph.

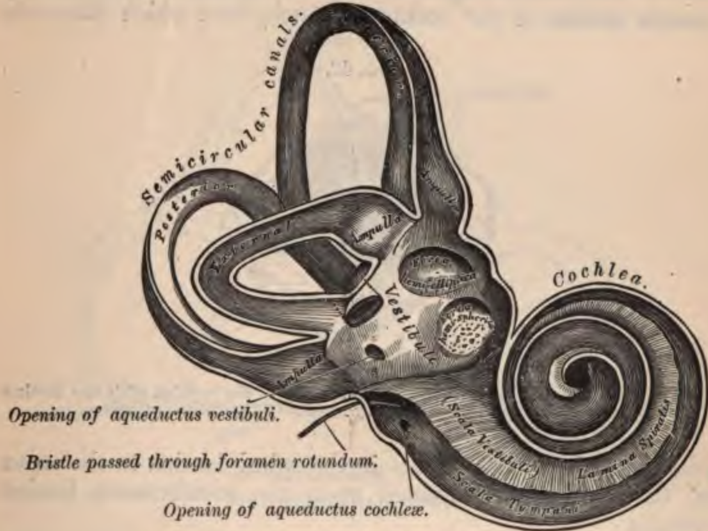
The Bony Labyrinth.

The Vestibule.—This cavity, which forms the greater part of the internal aural chamber, is an oval, bony space about one-quarter of an inch in diameter, and is situated immediately beyond the internal wall of the middle ear, with which it communicates in the live state through the fenestra ovalis. The anterior part of the inner wall of the vestibule presents above an oval depression, **fovea hemi-elliptica**, and below, a small round excavation, **fovea hemispherica**, into which filaments of the auditory nerve enter through minute foramina in the inner wall, while the posterior portion of the vestibular cavity reveals the **aquæductus vestibuli**, which passage connects with the subdural space. In front the vestibule becomes the **scala vestibuli of the cochlea** (Fig. 41), while posteriorly the **semicircular canals** communicate with the vestibule by five openings.

The Semicircular Canals.—Three small bony passages emerge from the posterior aspect of the vestibule, follow a nearly circular course through the osseous tissue, and terminate in a dilated portion, **ampulla**, where they again communicate with the cavity. One of the canals passes upward, and is termed the **superior semicircular canal**; another, the **posterior semicircular canal**, runs both vertically and backward; while the shortest of the three, the **external semicircular canal**, takes an outward and backward course. The former canals possess at one end a common opening into the vestibular chamber.

The Cochlea.—Situated antra-inferiorly to the vestibule, with which it communicates, and internally to the promontory which is formed by its first turn, a spiral canal, which resembles that of a minute snail-shell, is formed in the petrous bone, constituting the cochlea (Fig. 42). It consists of a conical central axis, the **modiolus**, around which a tapering spiral canal makes two and one-half turns, ending in an apex which points outward and forward, while the base of the cochlea faces the internal auditory meatus. Winding around the modiolus from the base to its apex, a thin bony plate, **lamina spiralis ossea**, projects from the central axis outward, midway across the lumen of the canal (Fig. 44), where it joins, on its lower edge, a **lamina spiralis membranosa** or **membrana basilaris** from the opposite side, thus dividing the spiral canal into two passages—the **scala vestibuli** above and the **scala tympani** inferiorly, which communicate at the apex of the cochlea

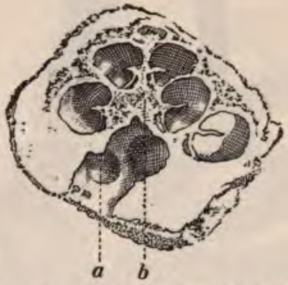
FIG. 41.



The osseous labyrinth laid open (right ear). (Gray.)

through a small foramen. The former passage enters the vestibule, while the latter communicates with the tympanum in the late stage through the fenestra rotundum, which is normally occupied by the membrana tympani secondaria. Through several canals in the base of the modiolus, blood-vessels and bundles of the cochlear branch of the auditory nerve are distributed to the scalæ. Within the **canalis spiralis modioli**, which tunnels around the modiolus near the junction of the bony lamina spiralis, rest the enlarged terminations, **ganglia spirala**, of the cochlear branch, from which filaments pass through minute canals in the spiral lamina to the **organ of Corti**, which is situated upon the membranous lamina spiralis.

FIG. 42.



Section of the osseous capsule and of the modiolus of the cochlea with the lamina spiralis ossea: *a*, internal auditory meatus; *b*, modiolus. (Politzer.)

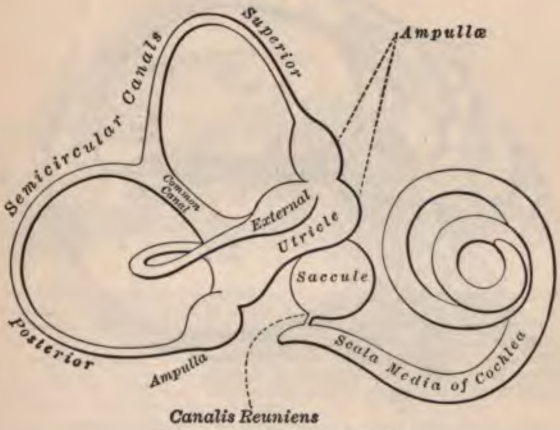
The Membranous Labyrinth.

As indicated previously, the membranous labyrinth consists of a series of fibrous pouches and tubes, filled with an endolymph, which form a loosely attached inner lining of the bony labyrinth, from the walls of which it is separated by an intervening perilymph. Hence, the outlines of the membranous labyrinth resemble those of the osseous portion (Fig. 43).

The Utricle.—This oblong sac forms the greater part of the vestibular portion of the labyrinth, and occupies the posterior part of the bony vestibule, forming an attachment to the inner wall in the fovea hemi-elliptica. Within the utricle at this point there is a **macula acustica** with its specialized **protoplasmic cilia,** which receive neural filaments from the auditory nerve. Posteriorly the utricular cavity communicates with the membranous semicircular canals.

The Saccule.—This globular pouch lies antra-inferiorly to the utricule in the **fovea hemispherica** near the mouth of the cochlea. A **macula acustica** is also present in the saccule at the point of its attachment in the hemispherical depression, from the bottom of which tiny nerve-filaments enter the macula. No communication is demonstrable between the utricle and the saccular cavity, though the latter joins that of the scala media through a minute passage, the **canalis reuniens.**

FIG. 43.

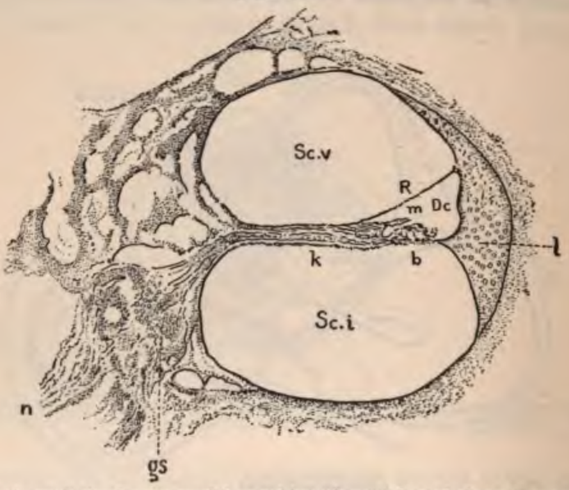


The membranous labyrinth.

The Membranous Semicircular Canals.—In number and shape these correspond to the bony canals which they line, and become continuous with the utricular cavity, as indicated in Fig. 43. In the **ampullæ** there occur the **cristæ acusticæ**, which are similar to the specialized areas of the utricle and saccule and also receive filaments from the auditory nerve.

The Scala Media or Ductus Cochlearis.—This triangular membranous passage, which traverses the entire length of the spiral canal of the cochlea, is a subdivision of the outer portion of the scala vestibuli, formed by the membranous spiral lamina or **membrana basilaris, ligamentum spirale,** and the **membrane of Reissner** (Fig. 44). Upon the basilar membrane rests the remarkable perceptive mechanism of Corti. In general the **organ of Corti,** as it is termed, consists of a ridge of polymorphous cells (Fig. 45), formed by two rows of modified columnar, epithelial cells, which, separated at the base, incline toward each other and interlock at the top, forming an archway within which a minute triangular pas-

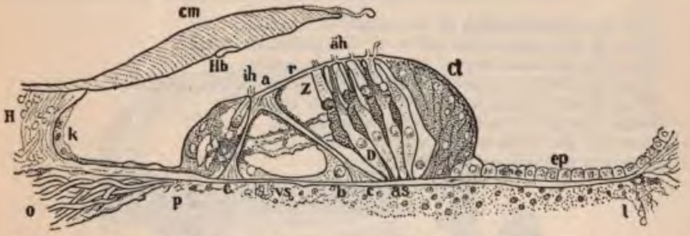
FIG. 44.



Section through the first turn of the cochlea of a newborn infant: *Sc.v*, scala vestibuli; *Sc.i*, scala tympani; *k*, lamina spiralis ossea; *b*, lamina basilaris; *l*, ligamentum spirale; *R*, membrana vestibularis; *Dc*, ramus cochlearis; *o*, Corti's organ; *m*, Corti's membrane; *n*, fasciculus of the ramus; *gs*, ganglia spirale. From a preparation in the author's collection. (Politzer.)

sage, **tunnel of Corti,** is enclosed. Upon either side of this epithelial arch rests a group of specialized neuro-epithelium, from the top of which minute cilia project into the lumen of the canal, which is filled with a thin fluid. Beyond this group the modified cells gradually merge into the epithelial layer of the membrana basilaris. From the ganglia spirala, tiny neural filaments may be traced to the ciliated epithelia through minute passages in the osseous spiral lamina. Extending from an epithelial thickening upon the upper edge of the bony lamina spiralis, a **membrana Corti** projects to the

FIG. 45.



Terminal filaments of the cochlear nerve, with Corti's organ, as found in the human subject: *o*, lamina spiralis ossea, with the nerve-bundle of the ramus cochlearis; *pl*, lamina spiralis membranacea; *H*, tooth of Huschke (crista spiralis); *C*, inner rods of Corti; *C''*, outer rods of Corti; *r*, lamina reticularis; *Z*, Corti's cells; *D*, Deiter's cells; *ih*, inner hair-cells; *oh*, four outer hair-cells; *e*, radiating tunnel fibres of the ramus cochlearis passing to the cells of Corti; *k*, cells of the sulcus spiralis interior; *Cl*, Hensen's supporting cells; *cm*, Corti's membrane; *vs*, vas spirale; *tr*, ligamentum spirale. (Politzer.)

ligamentum spirale of the outer wall, thus separating the organ of Corti from the rest of the scala media.

The Arteries of the Labyrinth.—The **vestibular** and **cochlear branches of the internal auditory artery** form the chief blood-supply of the internal ear, through anastomosis with the tympanic plexus through the internal wall of the tympanum, as previously quoted from Politzer.

The Auditory Nerve.—After a division in the internal auditory meatus the auditory nerve is distributed to the labyrinth through its two branches: the **vestibular nerve,** which through its three subdivisions sends filaments to the maculæ of the utricle, saccule, and semicircular canals; while the **cochlear nerve,** which traverses the modiolus, ends with the ganglia spiralis, from which a large number of minute filaments terminate in the ciliated cells of the organ of Corti.

Thus the sonorous vibrations imparted to the membrana tympani from without are transmitted across the tympanic cavity by the ossicular chain, which, impinging upon the fluid contents of the labyrinth, sets in harmonious motion the cilia of the organ of Corti, from which the impulse through the intervening auditory nerve reaches the auditory centre of the brain and is there interpreted as sound.

QUESTIONS.

Give the classification of the organ of hearing.
What is the general direction of the auditory canal?
Describe the anatomy of the tympanic cavity.
What important structures are associated with the mastoid?
What is the function of the Eustachian tube?
Name the subdivisions of the internal ear.
Describe the scala media and the organ of Corti.
Give the physiology of hearing.

CHAPTER II.

EXAMINATION OF THE PATIENT.

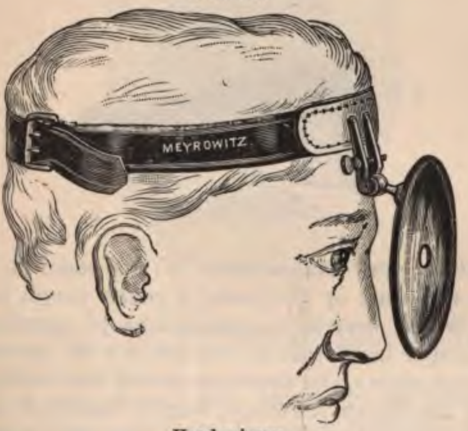
HISTORY OF THE AFFECTION.

WHEN a patient presents himself for consultation regarding an ear trouble, the problem which confronts the physician is the determination of the cause, location, and extent of the affection. The first procedure, then, should be the elicitation of a complete **history** as to the probable etiology, symptoms, and duration of the disease, remembering that inflammatory conditions of the nasopharynx, resulting from either local or systemic disorders, are the usual cause of aural disturbances. Specific information should be obtained as to the presence of impaired function, pain, tinnitus, discharge, and vertigo. *From these data* alone a diagnosis may frequently be made, but a thorough physical and functional examination by the following methods will frequently reveal the presence of an unsuspected condition. Too much emphasis can not be laid upon the importance of details in aural work, both in the examination and treatment of the conditions present, for therein only lies the secret of obtaining successful results.

PHYSICAL EXAMINATION.

Inspection of the External Parts.—In the examination of the **aural region** the physician should note the condition of the auricle, the orifice of the external auditory meatus, the parotid gland in front, and the mastoid process behind. Special attention should be directed to the latter, as it is always liable to be involved in inflammatory conditions of the tympanum. *A comparison of the part should be made with that of the opposite side, especially if malformations or swellings be suspected, or a tenderness of any portion be elicited by palpation.*

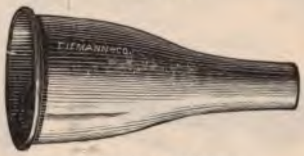
FIG. 46.



Head mirror.

Condition of the Meatus and Membrana Tympani.—To examine these structures properly requires a bright illumination, which may be either natural or artificial, though the latter is to be preferred because of its uniformity of light; a reflecting mirror (Fig. 46) attached to a headband so that the hands may be free to manipulate the auricle and the necessary instruments; and a speculum (Fig. 47) to separate the sides of the cartilaginous meatus, thus permitting the light which is reflected from the mirror to be directed into the canal. In the **introduction of the speculum** the curved direction of the meatus, as previously indicated, may be straightened, in the adult, by drawing the auricle upward, backward, and outward, while in the child the auricle should be pulled downward and backward, thus separating the upper and lower walls, which are membranous and frequently lie in contact.

FIG. 47.

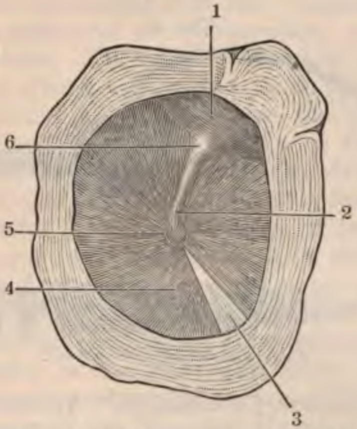


Gruber's speculum.

When instrumental examination of the canal is made with either the speculum or the probe, a reflex cough is sometimes produced, which may be so marked as to interfere seriously with the proper inspection of the interior of the meatus. As the speculum enters, the **examiner should note within the canal** the size of the lumen, its shape, length, condition of the lining epithelium, amount of cerumen, and the number and arrangement of the hairs, as all may be factors in the production or aggravation of a pathological condition within the meatus or even the tympanum.

Extending across the distal end of the canal will be seen the membrana tympani, which presents normally a pearly gray appearance. From its antra-superior aspect will be observed the faint outlines of the long handle of the malleus as it extends downward and backward between the outer dermal layer and the inner mucous covering of the membrane, terminating in a slight depression, umbo, at the centre (Fig. 48). At the upper extremity of the long handle, the short process, resembling in appearance a small pustule, is distinctly outlined, from which point the lower folds of the membrana flaccida may be followed anteriorly and posteriorly to the wall of the meatus. The cone of light, which occupies the antra-inferior quadrant of the drum membrane normally, may be displaced, distorted, or even absent, due usually to an inward bulging, retraction of the membrana tympani, resulting from an improper aëration of the tympanic cavity through a partial or complete occlusion of the Eustachian tube; though an outward curving of the membrane, resulting from the pressure of a fluid within the tympanum, may also cause the cone of light to disappear.

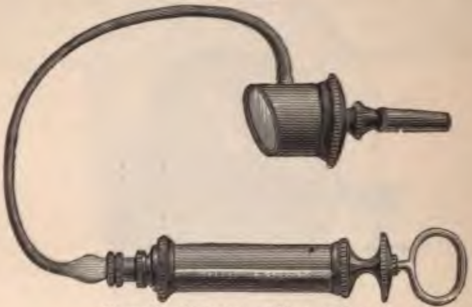
Fig. 48.



The normal membrana tympani (right ear): 1, membrana flaccida; 2, long handle; 3, cone of light; 4, membrana vibrans; 5, umbo; 6, short handle.

The **points in an inspection of the tympanic membrane** to be specially observed are its color, position, evenness of surface, and continuity of structure. The usual alterations in color are produced by an inflammatory condition within the meatus or tympanum, when the membrane assumes a tint ranging from a slight pinkish flush to that of an angry, dark, reddened, injected condition, while formations of atrophic areas, cicatrices, and calcareous deposits produce whitened areas in the membrane. The deviations of the normal position are traceable to either a tubal obstruction or tympanic adhesions to the inner wall of the tympanum, when the drum membrane is forced inward, or a fluid, serous or purulent, within the middle ear, when the membrane will bulge outward if the intratympanic pressure be sufficient. Atrophic areas, various deposits, ulcerations, polypi, and granulations, together with internal adhesions, are the common disturbing elements of the evenness of surface. An interruption in the continuity of structure results either from an inflammatory condition, perforation, or ulceration, a blow upon the auricle leading to rupture, or from a surgical procedure, incision, for therapeutic or exploratory purposes.

FIG. 49.



Siegel's otoscope, with rarefier.

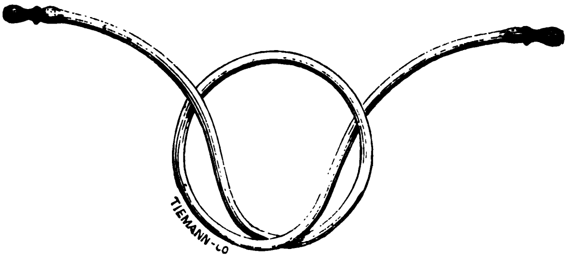
Atrophic areas and adhesions of the membrana tympani to intratympanic structures are diagnosed, in questionable instances, by the aid of the Siegel otoscope (Fig. 49), with which the air in the external auditory meatus may be rarefied, when it will be observed, while inspecting the drum head through the instrument, that with each movement of the piston in rarefaction and condensation there will be a corresponding outward and inward excursion of the whole membrane with an evenness of surface; but in the atrophic areas the bulging will be greater than that of the rest of the membrane; while with internal adhesions, areas will be seen where the mobility is restricted or lost, corresponding to the point of attachment.

Examination of the Nasopharynx.—As previously noted, the nasal and pharyngeal passages should be carefully examined in all aural disturbances where an inflammatory condition may exist, as an involvement of the tympanum frequently results from extension of an inflammatory process through the Eustachian tube. The respiration should be free through the nares, which often are narrowed by an hypertrophic condition of the turbinal mucous membrane, though the turbinate bone itself may be enlarged congenitally or through disease. Malformations of the septum, comprising deviations, spurs, thickenings, will also frequently sufficiently impede the influx of air to account for a chronic congestion, if not inflammation, of the mucous membrane in and about the pharyngeal orifice of the Eustachian tube. In an examination of the pharynx the usual etiological factors observed are an hypertrophic, granular or follicular pharyngitis, enlargement of the faucial tonsils, or adenoids in the pharyngeal vault. For details in the examination and treatment of these conditions, consult the volume on Diseases of the Nose and Throat of this series.

Examination of the Eustachian Tube.—While making the physical examination of the nasopharynx, the condition of the Eustachian orifice should be noted and the surrounding structures scrutinized for the presence of polypi, enlarged lymphatics, posterior septal and turbinal deviations, together with thick, tenacious discharges, any one or all of which are capable, as are also hypertrophic faucial tonsils, of mechanically obstructing the lumen of the pharyngeal meatus.

To determine the patency of the Eustachian tube the following methods are in vogue, by means of which air may be forced through the tube into the tympanic cavity, when, if the passage be partially or completely occluded, the function of audition will be correspondingly improved thereby, providing there be no organic lesions of the conducting or perceptive mechanisms. While employing any of these procedures, the physician should use the auscultation tube (Fig. 50), one end being placed in the examiner's external auditory meatus, while the other rests in the patient's meatus, by means of which additional information is gained as to the size of the lumen, presence of fluid, and the amount of pressure necessary to drive the air through the canal. By this method alone is the operator often certain that the entering air has reached the tympanum, when the impact of the air-current will be heard against the membrana tympani, and the resulting outward movement of the patient's drum head will produce an inward excursion of that of the examiner's, which he can easily feel.

FIG. 50.

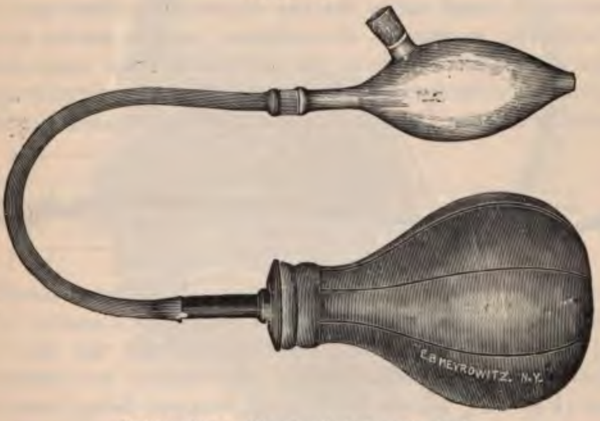


Auscultation tube.

The Valsalva Method.—This procedure consists of an attempt at forced expiration while the nose and mouth are held firmly closed, thus forcing the air through the passage of least resistance, the Eustachian tube. This method should be used only in determining the permeability of the tube or the mobility of the membrana tympani and ossicles, and should not be taught the patient to be used at will as a therapeutic means of relieving the stenosis of the canal, as the resulting congestion incident to its employment tends to aggravate an existing in- n of the tubal and tympanic mucous membrane. A relaxed condition of the membrana tympani or ossicles also not infrequently follows its repeated and forcible use.

The Politzer Method.—In using this mode of inflation the patient is directed to take a sip of water from a glass and retain it within the mouth until instructed by the physician by word or sign to swallow. The nozzle of a Politzer air-bag (Fig. 51) is placed snugly into one nostril, while the other is held firmly closed. The patient then swallows at the given signal, and the air-bag is simultaneously compressed, when, if the soft palate rests tightly enough against the pharyngeal wall, the air makes its passage up the Eustachian tube into the tympanum, as may be heard by the auscultation test. Instead of employing the act of swallowing to close the pharyngeal space, the patient may be instructed to forcibly pronounce the word "hook," prolonging the "k" sound, or markedly distend the cheeks in an attempt at forced expiration, when the air-bag may be used as previously indicated. The compression of the air-bag should always be guarded until the necessary force of inflation is learned, otherwise, if the tube

Fig. 51.

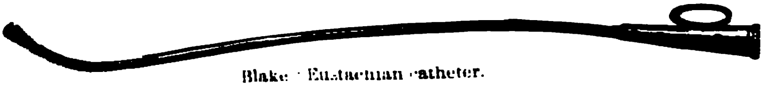


Politzer bag, with glass inhaler attached.

be patent and a strong compression made, the forcible entrance of the air into the tympanic cavity will be attended with great discomfort, if not actual pain, and frequently vertigo is thus momentarily produced by the sudden outward movement of the foot-plate of the stapes, which lowers the intralabyrinthine pressure. Rupture of the membrana tympani has resulted from the presence of these conditions, attended with atrophic areas in the drum membrane, which are, of course, points of weakened resistance.

The Principles of Catheterization of the Eustachian Tube.—Before attempting to employ the catheter, the operator should examine the nasal cavity for any obstructions that may interfere with the passage of the catheter; and to render the procedure less objectionable to the patient, a 4 per cent. solution of cocaine may be sprayed into the nostrils or preferably applied directly by means of a cotton-tipped applicator unless the patient has an idiosyncrasy against the drug. Whether a metallic or hard rubber catheter should be employed is a matter of personal choice and experience. For his own use the writer prefers the silver probe-pointed instrument as devised by Blake (Fig. 52), which permits of changes being made in its curvature by bending and its sterilization by means of steam. These catheters come in four different sizes, to fit the variously-sized orifices of the Eustachian tube, but No. 2 is the size most frequently employed.

FIG. 52.

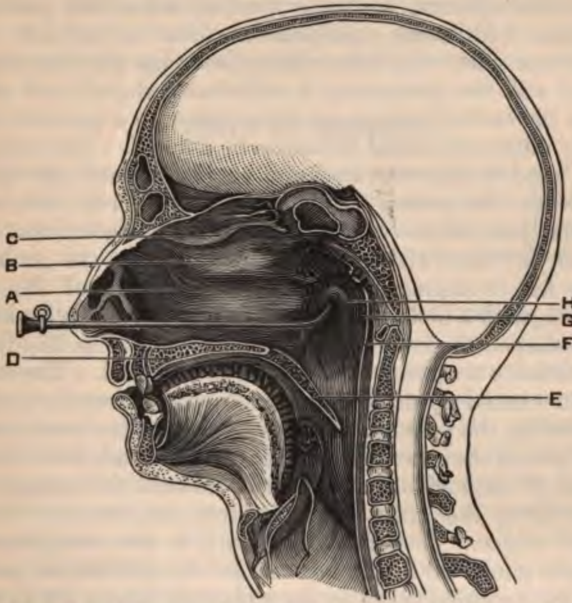


Blake's Eustachian catheter.

For the inflation of the tube after the catheter has been placed in the Eustachian orifice, an ordinary atomizer-bulb should be attached to the funnel-shaped end of the catheter, before its introduction, by means of a small flexible intervening rubber tube about one foot in length. Otherwise, if the bulb were connected after the passage of the catheter, the necessary manipulations would be liable to displace the catheter and discomfort the patient very much.

The Technique of Catheterization of the Eustachian Tube.—Seated or standing in front of the patient, the examiner places the fingers of the left hand upon the patient's forehead, while with the thumb the end of the nose is forced upward. With the other hand, while the patient is holding the bulb, the operator introduces the probe-pointed extremity into the nostril with the curved portion directed downward and resting upon the floor of the nasal cavity. The catheter is gently pushed along the inferior meatus until the curved extremity reaches the postpharyngeal wall, when the cath-

FIG. 53.



Vertical section of the nasopharynx, with the catheter introduced into the Eustachian tube: A, inferior turbinated bone; B, middle turbinated bone; C, superior turbinated bone; D, hard palate; E, velum palati; F, posterior pharyngeal wall; G, Rosenmüller's fossa; H, posterior lip of the orifice of the Eustachian tube. (Politzer.)

eter may be introduced into the Eustachian orifice by one of two courses: (1) It is now rotated directly inward and brought forward until the curved portion rests against the posterior end of the septum. The catheter is so turned that the probe end describes a semicircle, passing downward along the posterior surface of the soft palate and finally coming to a rest directly outward with the ring of the funnel end pointing toward the external auditory meatus, when the probe end will pass, with some manipulation, into the mouth of the Eustachian tube. (2) After the catheter has reached the pharyngeal wall the instrument is withdrawn slightly and rotated so that the curved end passes from below to a position almost directly outward toward the ear to be inflated. The extremity in the hand is then elevated and carried toward the opposite side, when the probe end will encounter the side of the pharynx just back of the Eustachian protuberance. The catheter is now withdrawn carefully, when the probe end will be felt to impinge against the posterior lip of the orifice, and then, with a little added traction, pass over the lip into the funnel-shaped orifice of the Eustachian tube (Fig. 53), imparting a definite sensation to the experienced hand. The catheter is again rotated upward so that the curved extremity points, as indicated by the position of the ring, toward the outer canthus of the eye on the corresponding side of the head, while the funnel end of the instrument is carried toward the opposite side, which forces the inner extremity into the lumen of the pharyngeal orifice. The connecting bulb is now compressed by the right hand, while the left retains the catheter in position. With the auscultation tube, the examiner listens for any diagnostic signs as the air passes through the tube into the tympanum.

As to which is the preferable method of inflation, Politzeration or catheterization, will depend wholly upon the condition of the nasal cavity and the lumen of the Eustachian tube, together with the experience of the operator with either mode. The advantages of the former are: its ease of application, its utility in obstructed nasal passages, nonliability of injuring the tubal or nasal mucous membrane, and its preferable use in children and nervous individuals. With catheterization, however, the following favorable points are observed: one ear only may be inflated; amount of force to open the tube can be definitely ascertained; the process may be repeated without annoyance to the patient; its success depends wholly upon the operator; medicated vapors and applications may be made directly to the tubal mucous membrane through the catheter, while slender bougies or probes may be introduced into the Eustachian canal thereby, affording an additional method of diagnosis and treatment of obstructions within the tube.

TESTS OF HEARING.

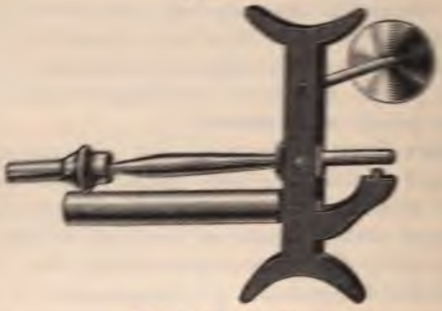
Having completed the physical examination of the aural structures, the physician should next determine carefully the functional condition of the organ of hearing, and record his findings for future reference in noting the course of the disease and the results of treatment.

Quantitative Tests of Hearing.—In determining how much the function of audition has been impaired, it is only necessary to ascertain at what distance a sound of given intensity and pitch is heard and compare it with that of the normal ear. While employing the various procedures, the patient's eyes should be closed to eliminate the imagination of hearing, and the organ not under the test should be stopped by the moistened finger of the patient or with a tight plug of cotton, so that only one side will perceive the sound.

As **impairment of hearing for conversation** is usually the first symptom recognized in aural disturbances, the examiner should note to what extent the hearing is affected in this direction by speaking in an ordinary or whispered tone of voice. It is well to employ short and varied phrases instead of sentences, as the patient will often construct the latter by catching a few of the words only, and it is equally important that the patient does not see the face of the speaker, as some individuals become very adept in reading the motion and position of the lips.

Impairment of Hearing for Sounds.—A watch or small clock may also be employed as a test by holding it beyond the normal distance of hearing and gradually approaching the ear until the ticking becomes audible. A stop-watch is preferable, as the test may be interrupted and thus trick the patient. As the pitch and loudness of the sound emitted by different watches or the human voice vary markedly, Politzer devised an instrument (Fig. 54) termed an **acoumeter**, which practically produces an uniformity of quality and intensity, by which means different observers may compare results; but, unfortunately, its intensity of click, which is heard at 45 feet by the normal ear, is too marked for the detection of slight alterations in function. In these instances the watch or tuning-fork offers a more delicate and reliable method of testing. In recording the findings of a quantitative test, a note may be made of the distance at which the instrument is heard; but preferably the results should be designated by a fractional term, which will indicate at a glance the amount of defect as compared with that of the normal ear. For instance, if the tick of a given watch be heard at 60 inches normally, and the patient perceives the sound at 30 inches, then the fraction $\frac{30}{60}$ would indicate definitely the state of his hearing, and would be understood by other observers in the same line of practice.

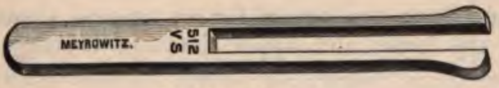
FIG. 54.



Politzer's acoumeter.

Qualitative Tests of Hearing.—In testing the quality of perception the physician will find that there is a quite definite range of hearing with the normal ear, which may be altered by diseased conditions of either the conducting or the perceptive apparatus. There is a point below which the ear can not hear lower placed sounds, while for the high tones a corresponding limit is reached beyond which the sounds become inaudible. These dividing-lines between audition and deafness are designated as the lower and upper limits of hearing respectively. By ascertaining their positions, which are normally placed at 16 and 32,500 tone-vibrations per second, and determining the duration of perception by air and bone conduction, the examiner is thereby enabled to decide whether the difficulty be located in the conducting or the perceptive mechanism, as these points are fairly constant in the normal state, but are characteristically altered in disorders of the middle or internal ear.

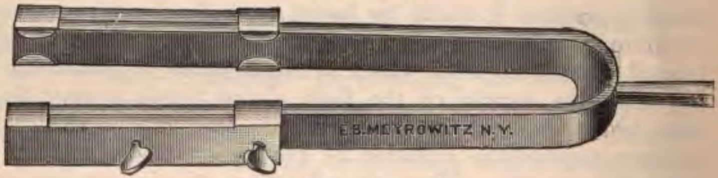
Fig. 55.



Blake's tuning-fork.

For instance, if a **Blake tuning-fork** (Fig. 55) making 512 single vibrations per second were held before the external auditory meatus, it would be heard normally for 30 seconds or more, providing a corner of the fork were not directed toward the canal, when the vibrations would be inaudible; but with the handle of the fork placed firmly against the mastoid bone, about one inch directly back of the external auditory meatus, the duration of perception would approximate 13 seconds. Thus the duration of air conduction is normally about twice that of bone transmission. This procedure has been named the **Rinne test**, in honor of the scientist who first observed the above relationship of air and bone conduction. When the vibrating-fork is held before the meatus, the vibrations are imparted through the intervening air to the membrana tympani, and thence to the labyrinth through the ossicular chain. Hence, if there be any obstruction to its freedom of passage—*e. g.*, a foreign substance occluding the external auditory meatus, or a restriction of the mobility of the drum head or the ossicles by the presence of adhesions or a fluid within the tympanum—the duration of hearing by air conduction will be correspondingly diminished. With the handle directed against the mastoid bone, the sound-vibrations of the instrument reach the perceptive organ through the substance of the bone, thus eliminating the conducting apparatus; and if the nervous mechanism of the labyrinth, the auditory centre of the brain, or the connecting auditory nerve be affected, the duration of bone conduction will be limited, the amount depending upon the extent of the lesion. Occasionally in hyperæsthetic conditions of the nervous system the duration and range of audition appears greater than normally observed. Theoretically the duration of air and bone conduction for all musical sounds present between the lower and upper limits of hearing should be ascertained in a complete test; but, practically, tuning-forks tuned to C, making 128, 256, 512, 1024, and 2048 single vibrations per second respectively, furnish a very complete diagnosis. The lower limit is tested by means of a large tuning-fork (Fig. 56), which records 26 to 64 v. s. per second, while the upper limit is determined with sufficient accuracy by the use of the **Galton whistle** (Fig. 57), which is capable of giving from 3500 to theoretically 84,000 v. s. per *second, though* the König rods give the more complete test,

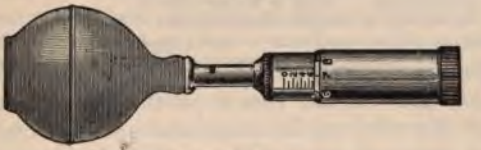
FIG. 56.



Denche's large tuning-fork.

but require too much time for routine use. Another phenomenon of aural perception has been designated as the **Weber test,** which is observed with the handle of a vibrating fork resting upon the skull in the median line, either upon the vertex, forehead, or the maxillary region, when, if the external auditory meatus be closed by the finger, the sound will be heard more distinctly upon the corresponding side. Thus, clinically, if there be an impairment of the hearing in one ear only, or the two sides be unequally affected and the patient recognizes the vibrations as louder in the deafened ear, these facts indicate that an obstruction exists in the conducting apparatus of the same side, which may be a foreign body in the meatus, adhesions of the membrana tympani or ossicles, or fluid within the tympanum if the Eustachian tube be patent. Were the sound heard more distinctly upon the better side, this would point to an impairment of the perceptive mechanism of the more deafened ear. This method, therefore, should precede the Rinné test in a systematic examination of the hearing.

FIG. 57.



Galton's whistle.

In general, then, with lesions of the conducting apparatus, the duration of air conduction is shortened (bone transmission remaining normal), while the lower tone limit is raised. In affections of the internal ear, a diminution of duration for both air and bone conduction is shown, but the latter is relatively more reduced, while the upper limit is lowered, its amount depending upon the extent of the alterations in the labyrinthine nervous mechanism.

From the data obtained in the physical and functional examination of the aural structures, the cause, location, and extent of the affection may be determined, when the physician will be in position to consider properly the methods of treatment.

QUESTIONS.

What points are to be elicited in the history of a case?
What structures are inspected in a physical examination?
Describe the appearance of a normal membrana tympani.
What points should be observed in its inspection?
Describe the Politzer method of inflation.
Give the technique and advantages of catheterization.
Name the different methods of testing the hearing.
What is meant by the lower and upper limits of hearing?
What is Rinné's test? What is Weber's test?
What is the utility of the qualitative tests?
What instruments are necessary to make these tests?
What are the characteristic alterations indicative of involvement of the middle ear? Of the labyrinth?

CHAPTER III.

DISEASES OF THE AURICLE AND EXTERNAL AUDITORY MEATUS.

MALFORMATIONS.

Significance.—Occasionally among those who present themselves for consultation regarding aural troubles, the physician will note instances of congenital defects in the auricle or the external auditory meatus, which may exist without the knowledge of the patient, especially when the malformation is not marked. These freaks of Nature are sometimes associated with perversions of mind and nervous system, and are thus looked upon by many observers as stigmata of degeneration. While it is true that many cases occur apparently substantiating this belief, the most aggravated forms of malformation may exist without any signs of impaired mentality.

Common Abnormalities of the Auricle.—Among the usual deviations from normality may be mentioned the excessively large or small auricle, abnormality of position or attachment, *supernumerary* ridges or fossæ, deformities or absence of the tragus, rudimentary or cleft lobule, indentations of the helix, and mixed conditions.

Common malformations of the meatus frequently appear with those of the auricle, especially when the latter involve the structures about the orifice of the canal. Exostoses in the bony portion, cartilaginous spurs in the outer meatal passage, very small lumen (atresia)—which may be round, but is usually slit-shaped—or even complete stoppage or absence of the canal are conditions that may be occasionally met. Of course, when the malformations markedly diminish the lumen of the meatus, deafness of various degrees will add to the discomfort of the individual.

Treatment.—Regarding the advisability of surgical procedures for cosmetic effects, it may be said that much may be accomplished for the amelioration of the deformity by skilfully executed plastic operations which have for their purpose the removal of superfluous structures. As to the conditions of atresia and absence of the meatus, it is a generally accepted fact that surgical interference is often disappointing on account of the formation of cicatrices and granulation tissue, which tend to undo an otherwise brilliant result. These cases, therefore, should be referred to the specialist for treatment.

SEBACEOUS CYSTS.

Synonyms.—Steroma; Wen; Atheroma.

Definition.—A sebaceous cyst is a noninflammatory, rounded—either soft or hard—elastic tumor, varying in size, which is produced by the distention of a sebaceous gland by its retained secretions.

Etiology.—Irritation of the mouth or duct of the gland from mechanical or inflammatory sources, thickened secretions which can not be discharged from the gland, and hypersecretion.

Symptoms.—The tumor is usually located in the lobule, though it may occur at any point upon the surface of the auricle. In the external auditory meatus the cysts develop in the cartilaginous portion, where the glandular structures are usually confined. The tumor gradually enlarges without pain or discomfort unless appearing in the bony portion of the canal, which is rare. Attaining a certain size, it frequently remains stationary in its growth for an indefinite period unless irritated, when the secretion is liable to be increased. The only interference with the function of the ear occurs when the tumor attains such a size in the meatus as nearly or completely to occlude the lumen, producing varying degrees of deafness, which occasionally is altered by movement of the jaws, when the lumen of the meatus is changed.

Treatment.—After producing anæsthesia of the part by means of Schleick's injections or any other local anæsthetic, the tumor is removed by a careful dissection so that the sac or distended gland is not ruptured, otherwise its complete removal will be difficult, and unless this is accomplished the cyst is liable to recur. A less preferable method is the incision of the sac, evacuation of the contents, and destruction of the secreting walls by means of caustics.

HÆMATOMA AURIS.

Synonyms.—Athæmatoma; Blood tumor.

Definition.—Hæmatoma auris is a bluish-red swelling of the auricle, due to an effusion of blood between the cartilage and the perichondrium.

Etiology.—This disorder is frequently the result of a blow upon the auricle or pulling of the ear, which ruptures a blood-vessel or separates the cartilage from the perichondrium, into which space the blood flows. Spontaneous rupture of atheromatous arteries, formation of fissures or cavities in the cartilages, proliferation of bloodvessels, and new growths are also recognized as etiological factors.

Symptoms.—Following a traumatism of the auricle, or occurring spontaneously, a tumefaction suddenly makes its appearance upon either side of the auricle, although it usually occurs upon the anterior surface, attended by a painful sensa- on at the site of the swelling and a feeling of warmth. The n is sharp and stabbing in character, due to the forcible separation of the tissues by the sanguineous effusion. On account of its frequent occurrence among the insane, it is claimed by some authorities that instances of spontaneity are traceable to local trophic changes, resulting from intercranial lesions. Others attribute its appearance in the demented to ill treatment or injury of the part by the attendants. The contents of the swelling frequently disappear by absorption, but may become purulent in character, attended with inflammatory symptoms. Should the swelling extend to or involve the meatus sufficiently, the hearing will be lowered from the occlusion of the canal, although this is unusual.

Treatment.—In order to reduce the swelling, cold soothing compresses may be applied for a time, when, if the tumefaction does not abate, an incision should be made at the most prominent point and the contents evacuated, following strict antiseptic procedures in the after-treatment. If the tumor be small, resolution may occur without interference. The patient should be warned that deformity of the auricle is liable to result from the cicatricial contractions consequent upon this affection, especially if the effusion of blood has been extensive.

PERICHONDRITIS AURICULÆ.

Definition.—An acute inflammation of the perichondrium.

Etiology.—May follow hæmatoma auris as a complication; often results from traumatism or frost-bites of the auricle.

Symptoms.—With the gradual appearance of an inflammation of the auricle, a bright-red swelling, which may occur at any point, slowly makes its appearance, attended with severe pain and a feeling of heat in the affected area. The serous effusion between the cartilage and the perichondrium, which accounts for the enlargement, may become so extensive as to involve the entire auricle, obliterating the ridges and fossæ as the perichondrium is dissected from the cartilaginous framework. Later the serous contents of the tumor may become purulent in character, which adds to the gravity of the condition. If left to itself, spontaneous evacuation of the fluid will result from perforation, followed with great deformity. The differential diagnosis of perichondritis from hæmatoma rests upon the fact that in the latter the swelling which results from an effusion of blood appears suddenly and unattended with inflammatory conditions, while the perichondritic exudate is serous and produces its enlargement gradually, following a previous inflammatory state.

Treatment.—Practically that of hæmatoma auris.

ECZEMA.

Synonyms.—Salt rheum ; Moist tetter ; Milk crust ; Scald.

Definition.—Eczema is an acute or chronic, multiform, inflammatory disease of the skin, characterized by the formation of vesicles, papules, or pustules attended with infiltration and thickening of the epidermis, and terminating in a desquamation or a seropurulent discharge with the formation of crusts.

Etiology.—The causes of this dermatitis are : (1) predisposing constitutional disorders, among which may be mentioned rheumatic tendencies, dyspepsia, constipation, mental and physical exhaustion, and the neuroses of functional or organic origin ; and (2) local causes producing irritation of the skin, such as excessive heat or cold, strong soaps, acids, alkalies, injuries of the skin, which in the case of involvement of the external auditory canal frequently results from foreign bodies being introduced into the meatus with the intention of scratching the skin or removing the ear wax, inspissated or impacted cerumen, and irritation by a chronic discharge from the tympanum.

Symptoms.—The most pronounced symptom is the itching, pricking, or burning sensation which first calls attention to the part, congestion of the region affected, presence of an exudate, formation of crusts, thickening of the skin, development of fissures, and desquamation. If the meatus be affected, the hearing may be altered mechanically by the presence of crusts, discharges, desquamations, or swelling of the skin, which might obstruct the lumen of the canal sufficiently.

Treatment.—In general remove the local and predisposing systemic causes, advise hygienic and dietetic measures, apply locally soothing and protective medication in the acute form, and stimulating ointments in the chronic state. Removal of the crusts is best accomplished by an oily preparation. For details of treatment consult a work on diseases of the skin, as eczema in this region does not differ from that affecting any other part of the body. Care should be exercised that the membrana tympani be not injured by either the medication employed or the instruments used in its application.

DIFFUSE EXTERNAL OTITIS.

Occurrence.—This form of inflammation of the external auditory meatus may occur either as an acute or chronic affection which involves, as its name implies, the greater portion, if not the whole, of the meatal lining membrane, gradually merging into the surrounding epithelium.

Synonyms.—Otitis externa diffusa acuta; Diffuse inflammation of the external ear.

Etiology.—As an idiopathic affection, the disease is rare. Usually it results from pathogenic microbic infection which enters through abrasions of the skin produced by traumatic influences, foreign bodies introduced into the canal, irritation by discharges from the tympanic cavity, or the instillation of improper fluids into the meatus, and secondary to furuncular inflammations of the epithelium.

Symptoms.—The symptoms are especially marked when the disease involves the osseous portion of the canal and the surface of the membrana tympani. Beginning with an itching sensation, a violent, radiating pain soon follows, which is increased by movements of the jaws, manipulation of the auricle, and lying upon the affected side. Tinnitus and deafness may supervene. The inflammation first makes its appearance as a congestion of the cutis, followed by marked swelling and infiltration; development of a serous or purulent discharge which softens the cuticle; exfoliation of the whitened, macerated dermal layers, leaving an angry, excoriated surface which is extremely sensitive; ulcerations of the membrana tympani may develop and result in perforation; granulations may spring from an ulcerative process of the meatal wall; or necrosis of the bony meatus may affect the unfortunate individual. In the chronic form there may occur an itching of the skin, slight degree of pain, varying amount of discharge, and desquamation which may obstruct the canal by the exfoliated débris, attended with a lowering of the aural function. Reflex cough is also thereby occasionally produced.

Diagnosis.—Inspection of the meatus and membrana tympani reveals the swollen, infiltrated condition of the skin, the discharge, and the whitened, exfoliating layers of the epithelium, which microscopically show the presence of micrococci or aspergillus fungus. This condition might be confounded with that resulting from a purulent discharge from the tympanum, but in the latter instance inspection would reveal the presence of a perforation of the drum head, which would indicate the nature of the affection.

Prognosis.—In the idiopathic cases the inflammation subsides in the course of a few days, while in those due to a traumatic origin the resolution is usually delayed because of the complicating ulcerations and attending granulations, which are not only slow in healing, but tend to the formation of strictures and atresia of the canal.

Treatment.—In the acute form, if the inflammation be mild, antiseptic and protective measures will usually suffice; while if the inflammatory conditions be marked, the patient should be put to bed, a saline purgative given, a hypodermic administered if necessary for the relief of the pain, the canal syringed with warm solutions of boric acid (saturated), bichloride of mercury (1:8,000), or carbolic acid (1:40), artificial leech and cupping applied in front of the tragus or immediately behind the auricle as is indicated, and the application of an ice-bag or coil to the mastoid. If the swelling does not abate within a day or so, scarification or incision of the infiltrated tissues extending down to the bone will prove beneficial, especially in the early stage. After the canal has been dried insufflations of boric acid powder are *recommended.* In the chronic form, after cleansing the canal with antiseptic solutions, applications of nitrate of silver solution (1 : 24 or stronger), instillation of boric-alcohol (1 : 20), or diachylon ointment may be used. If the alcoholic solution does not remove the granulations, they may be destroyed by cauterization, either with solid nitrate of silver, chromic acid, or preferably the electrocautery, the parts having been previously anæsthetized by the use of powdered cocaine.

ACUTE CIRCUMSCRIBED EXTERNAL OTITIS.

Synonyms.—Otitis externa circumscripta acuta; Furuncle of the external auditory meatus.

Etiology.—It will be recalled that the cartilaginous portion of the canal contains sebaceous and ceruminal glands, together with hair-follicles, which are liable to infection, thus producing the furuncle or boil within the meatus. The pathological micro-organisms gain entrance through abrasions or irritation of the skin produced by various agencies. Irritation from foreign bodies, instillations of caustic substances, chronic eczema, diffuse external otitis, and predisposing systemic disorders, such as debility, dyspepsia, anæmia, and diabetes, are also mentioned as rendering the patient susceptible to the affection. Frequently idiopathic instances occur in the most robust constitutions. Climatic conditions seem to exert an etiological influence in the production of this disease, as it occurs more frequently during the spring and autumn months. It is more commonly observed in adults than in children.

Symptoms.—The first symptoms that the patient may notice will usually appear as an itching or fulness in the meatus, which gradually increases, attended with the development of a severe, radiating pain, which is increased by movement of the jaws, so that mastication may become impossible; pressure upon the auricle also accentuates the trouble. The pain may be so severe as to prevent sleeping; the appetite may be deranged; debility and headache, together with an elevation of temperature, may be recorded. If the tumefaction produces a stenosis of the canal, tinnitus and deafness may ensue from the mechanical obstruction of the passage or the induced congest of the tympanum and labyrinth. The cervical, parotid, and preauricular glands may become increased in size from an extending infiltration; and occasionally, if the inflammatory area be situated upon the posterior wall of the canal, an œdematous condition may appear upon the mastoid immediately behind the auricle, which may be diagnosed a mastoiditis by the inexperienced. Within the meatus it may be observed that the tumor or swelling appears flattened, poorly defined, and slightly congested when the abscess is located deep beneath the surface; but if developed superficially, the furuncle usually presents a rounded, elevated, reddened tumor, which nearly or completely obstructs the canal, so that it may be impossible to see the drum membrane. After four or five days the contents of the tumefaction become purulent, and may rupture the surrounding structures and allow the discharge to escape, when the patient becomes more comfortable with the resulting diminution of the swelling and pain. Relapses are liable to recur from stoppage of the perforation through contraction or granulation growths. Sometimes several furuncles succeed each other in development or may appear at the same time. Occasionally infection is conveyed to the tympanum, and a resulting inflammation of the middle ear adds to the discomfort of the unfortunate sufferer.

Diagnosis.—As a rule this is not attended with any difficulty when the subjective symptoms, together with the signs of inspection, are carefully considered. Exostoses of the meatus, covered by an injected epithelium or bulging of the posterior wall incident to inflammation of the mastoid, might be regarded as furuncular swellings; but the judicious use of the probe will aid in the localization of the trouble.

Prognosis.—The termination of furunculosis of the meatus is favorable in uncomplicated cases. Under proper treatment the symptoms disappear within the course of a few days, when resolution is complete.

Treatment.—As the pain is the most aggravating symptom produced by the infiltration and distention of the tissues, the canal should be syringed with a warm antiseptic solution, preferably carbolic acid (1 : 40), which will partially anæsthetize the skin and prevent reinfection, followed by a deep incision into the most prominent portion of the swelling, whether suppuration be suspected or not. If a purulent focus has not formed, the escape of the serosanguineous fluid relieves the pain and feeling of tension in the meatus. Should the knife fail to reach the pus, it will soon make its way to the incision through the path of least resistance. Occasionally, after spontaneous rupture has occurred, especially if the amount of discharge has been small, it is advisable to enlarge the opening, which may be occluded by inspissated suppurative débris or the core of the furuncle. With the advent of the purulent discharge, the canal should be kept clean by means of warm antiseptic solutions, which will prevent infection of other portions of the meatus. After drying the canal with a pledget of cotton upon an applicator, a light packing of antiseptic gauze may be introduced into the canal as a means of protection and drainage. This procedure should be repeated as often as occasion demands. Applications of ichthyol solution (1 : 1), carbolic-glycerine (1 : 30), boric-alcohol (1 : 20), nitrate of silver (1 : 24), and hydrogen peroxide are recommended to be employed in the after-treatment; but the treatment outlined above has proved the most satisfactory in the hands of the author.

Diphtheritic External Otitis.—This rare condition of the meatus usually **occurs** as a complication of faucial diphtheria, and its **treatment** is that of the general disease, by means of antitoxin, together with antiseptic and protective measures locally.

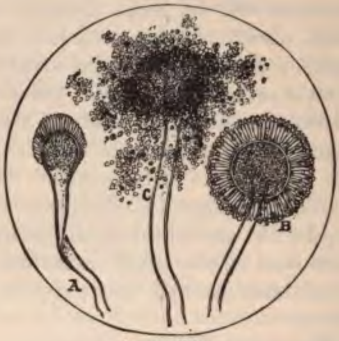
OTOMYCOSIS.

Occurrence.—Frequently, among foreigners who live under poor hygienic and dietetic conditions, this parasitic inflammation of the external auditory canal is found to exist. It is most commonly **caused by** *Aspergillus nigricans* or *Aspergillus flavescens*, which **produces** a slight superficial inflammation of the meatal lining membrane, attended by **symptoms** of itching, pain, desquamation, and, at times, tinnitus and deafness. The **treatment** consists of enforced hygienic measures, removal of scaly débris, and application of antiseptic solutions or powders.

SYPHILIS OF THE MEATUS.

Usually being a local manifestation of a general disorder, syphilis in this region **appears as** condylomata, ulcerations, and gummata. The condition last mentioned is rarely confined to the canal, but involves the tympanic cavity and labyrinth as well. Ulcerations in the meatus, especially if deep, may produce stricture of the passage, with resulting involvement of the hearing.

FIG. 58.



Aspergillus.

The **treatment** consists of the constitutional administration of mercury and potassium iodide, and the local use of antiseptic solutions employed as a douche, followed by applications of iodoform, boric acid, or calomel powders.

IMPACTED CERUMEN.

Significance.—While this disorder does not differ essentially in its symptomatology and treatment from that presented by *the presence* of other foreign bodies within the external auditory meatus, for the purpose of lucidity it seems advisable to consider this affection under a separate heading, as it obtains its origin in a perversion of secretion and retention within the canal.

Synonyms.—Inspissated cerumen; Ceruminous plugs.

Definition.—Impacted cerumen is a collection of an abnormal amount of ear-wax, mixed with various kinds of débris, which may partially or completely occlude the external auditory meatus.

Etiology.—The normal ceruminal secretion, which is the product of the ceruminous and sebaceous glands which are present in the cartilaginous portion of the canal, is under normal conditions removed from the meatus by the movements of the inferior maxillary and various manual procedures. Part of the secretion, however, is occasionally retained within the canal by various obstructions, when a nucleus is formed about which the cerumen gathers until the passage is nearly, if not entirely, closed. Among the many causal factors that may be instrumental in the production of ceruminal plugs may be mentioned: inspissated secretion from the tympanum; habitual or recurring hyperæmic conditions of the meatal lining membrane, which stimulate the glandular structures to hypersecretion; congenital or acquired strictures of the meatus, which tend to retain the normal or perverted secretions within the canal; a thickened condition of the ceruminous matter; the desquamations of eczema, diffuse external otitis, and other scaly conditions of the meatus; and various kinds of foreign bodies within the meatus.

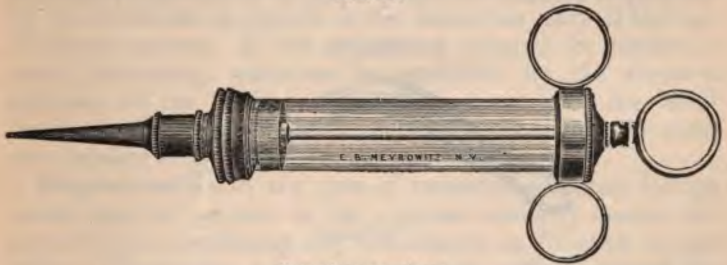
Symptoms.—It is frequently observed that large collections of cerumen may remain within the external auditory meatus for many years without any inconvenience to the patient so long as the plug does not rest against the membrana tympani and a passage is preserved between the cerumen and the meatal wall for the transmission of the sound-waves. With occlusion of the canal a sense of fulness is experienced; tinnitus and deafness of varying degrees may ensue; itching, irritation, and even a piercing pain may be produced by the hardened mass, especially when the jaws are moved from side to side; condition of apathy, resonance of patient's voice, nausea and vomiting, blepharospasm, and reflex cough are also occasionally met. Patients will frequently make the statement that the dulness of hearing appeared suddenly, which may be explained by the fact that the plug of cerumen was displaced against the membrana tympani by an unusual movement of the head or the jaws, or the impacted débris became swollen through the imbibition of fluids intentionally or accidentally introduced into the canal.

Diagnosis.—The subjective symptoms will often appear vague and indefinite, or of such a character as to mislead the physician; but examination with the speculum will reveal the presence of a ceruminal mass, which may appear a light yellow or dark brown in color. By means of a probe its consistency may be determined, which may vary from a soft greasy mass to a hard inspissated plug. Frequently it will be observed that the centre of the impacted cerumen is solid, possibly a foreign body, around which a softened layer has formed. Exostoses of the meatus covered with cerumen, inspissated purulent secretions from the tympanum, foreign bodies of cotton or paper which have become darkened with age, and dried clots of blood may be mistaken for a ceruminal mass.

Prognosis.—In uncomplicated cases careful removal of the impacted cerumen will be attended with complete restoration of the function and disappearance of the symptoms; but a guarded prognosis should always be made, as coincident affections of the membrana tympani, tympanum, or labyrinth may be present, which after removal of the débris may be attributed to the manipulations of the physician.

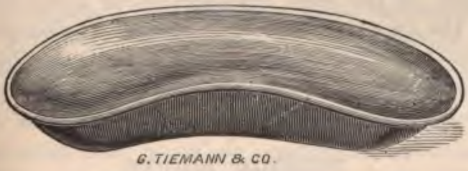
Treatment.—The most effective method of removing the ceruminal mass is by means of a forcible syringing of the meatus with a warm alkaline antiseptic solution, which procedure is not liable to injure the parts nor cause much discomfort, while the antiseptic solution will lessen the danger of infection of the tympanum should a previous or resulting perforation of the drum membrane exist. The current should be directed between the meatal wall and the impacted cerumen, so that the returning stream will aid in the expulsion of the mass. When the plug is hard, it may be necessary to continue the syringing for some time, as it requires an imbibition of the fluid to soften the matter sufficiently for its removal. The questionable procedure of advising the patient to make repeated instillations of an oily or alkaline solution into the canal for several days, with the intention of softening the débris before attempting its removal by means of the syringe, should not be employed, as the resulting swelling of the inspissated cerumen may damage the surrounding structures, and infection may be carried to the tympanum, through a perforation, by the instilled fluid. To effect properly the removal of the mass requires the use of a head-mirror to direct the light into the canal, a large syringe (Fig. 59), a pus basin (Fig. 60) or other receptacle, placed against the neck, under the auricle, to catch the returning

FIG. 59.



Bacon's ear syringe.

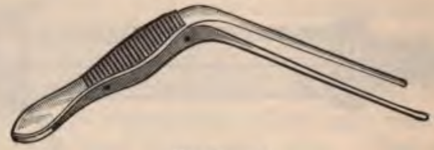
FIG. 60.



Pus basin.

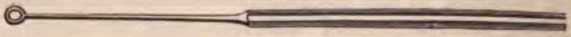
flow, and a towel or large napkin placed upon the patient's shoulder and within the collar to protect the clothing. The solution to be used in the syringing should not be contaminated by that which has been previously employed, especially if a perforation be suspected, but should be contained in a separate basin. Occasionally it may facilitate the removal of the plug of cerumen to employ a pair of ear forceps (Fig. 61) or small curettes (Fig. 62); but great care should be exercised that the meatal wall or the membrana tympani be not injured thereby. After the canal has been thoroughly cleaned, if there exists any irritation of the skin insufflations of boric acid may be made, and the meatus closed by a pledget of absorbent cotton, which should remain in the passage for a day or so to prevent the development of an inflammation within the canal or tympanum from undue exposure.

FIG. 61.



Ear forceps.

FIG. 62.



Ear curette.

FOREIGN BODIES.

Etiology.—From time immemorial there seems to have existed an inherent tendency in children to insert all objects, whose size would permit, into the mouth, nostril, and external auditory meatus. In a playful or spiteful mood they frequently perform this operation upon each other, and submit to the procedure with mingled joy and anticipation until a resulting discomfort terminates the act. Buttons, beads, pebbles, seeds of various kinds, such as peas, beans, wheat-kernels and coffee-beans, pellets of paper, cotton and leaves, pieces of matches, tooth-picks, and pencils are some of the objects that find their way into the youthful meatus. Insects of various varieties frequently enter the canal, with or without the knowledge of the patient, and become imprisoned by the ceruminal secretions or the numerous hairs of the cartilaginous meatus. If the membrana tympani be reached, a most distressing sensation is produced by the frequent attempts of the insect to effect its escape. In the adult foreign bodies usually obtain their presence within the auditory passage through accident or malicious intent.

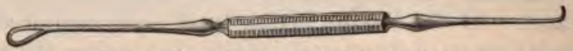
Symptoms.—As in the case of impacted cerumen, foreign bodies may be present in the external auditory meatus for years without producing any disturbance in function or sensation. Frequently a layer of cerumen gathers around the object, thus increasing its occlusion of the canal, and when this is sufficiently accomplished various symptoms make their appearance. If the foreign matter be of such magnitude and irregularity as to obstruct and irritate the meatus, the patient will complain of a sensation of fulness and pain, attended with tinnitus and an interference of hearing. Should the foreign body be a piece of wood, a bean, or any other substance which would swell in the presence of moisture, the resulting enlargement would be productive of injury of the surrounding structures and occasion a great amount of suffering. Various reflex phenomena may be produced through irritation of the trifacial and vagus nerves which distribute filaments to the meatal walls. Persistent cough, nausea and vomiting, epileptic attacks, and neuralgic conditions have been traced to this source, and relieved by removal of the foreign body from the canal. With a sufficient irritation the lining membrane of the meatus becomes swollen, ulcerated, and extremely painful. Granulations may develop, which, together with the œdematous condition of the meatal structures, may completely close the canal and hide the foreign body from view, although a purulent discharge may escape from the lumen. Secondary involvements of the tympanum, labyrinth, mastoid, and meninges from an extension of the inflammation have been reported.

Diagnosis.—By means of the speculum and a guarded use of a probe the nature of the obstructing body can easily be determined. Its consistence, position, size, and form should be noted, as these features will demand attention in effecting its removal. When the offending body is situated deep within the canal, and especially if the parts be swollen, it will often be difficult to ascertain these facts.

Prognosis.—While in the majority of instances the presence of a foreign body in the meatus and its removal are not attended with any special annoyance or danger to the function of hearing, it is well for the physician to be reserved in his statements, as there is no certainty of the conditions existing beyond the obstruction, especially if the object has been in the canal for some time and the lumen be occluded.

Treatment.—The method of removing a foreign body from the meatus will depend upon the consistency, location, size, and form of the substance. If it be not liable to enlarge by the absorption of fluids, the most pleasant and effective procedure will be a syringing of the passage with a warm alkaline antiseptic solution in the same manner as previously indicated in the removal of impacted cerumen. If the parts be sufficiently swollen to prevent its extraction, the canal should be kept as clean as possible by antiseptic irrigation and cold applications applied to the auricle or mastoid with a view to reducing the inflammation before making further attempts at displacing the offending body. Should the syringe fail to effect its expulsion, various instrumental procedures may be tried by means of forceps, loops of wire, or hooks deftly placed around and behind the substance with the intention of rolling or pulling it outward. *ution should* be exercised that the foreign body be not acci-

FIG. 63.



Hook and loop for removal of foreign bodies.

dentally forced inward instead, which would greatly complicate the condition.

If the removal be not accomplished by any of these methods, operative interference offers the only means of extraction. No definite rule can be given to guide the physician in determining at what stage recourse should be made to the knife; but should palliative treatment fail to check or improve the conditions present, the attendant is justified in advising an operation. This procedure consists of a series of incisions whereby the posterior part of the auricle and cartilaginous wall is separated from its attachments, and also at its junction with the posterior wall of the osseous portion, so that the posterior wall of the cartilaginous canal may be drawn outward and forward, when instrumental delivery of the foreign body will be facilitated. If the body be impacted in the bony portion of the meatus, it may be necessary to chisel away part of its posterior wall to reach the imbedded obstruction.

When an insect enters the canal, it is best to terminate its tormenting struggles for liberation by instillations of an oily preparation, which will not only kill the intruder and form a protective to the irritated lining membrane, but also facilitate its removal by syringing. The larvæ of blue-bottle flies are sometimes found in the meatus, especially in cases of neglected, offensive, purulent discharges from the tympanum or suppurative conditions of the external auditory meatus. If they reach the tympanic cavity through a perforation, it is difficult to dislodge them; but the procedure outlined above will finally effect their displacement, especially if a few drops of petroleum or turpentine be added to the instillations, which will cause them to migrate from their abode out into the canal, from which they can be easily removed.

QUESTIONS.

What are the common affections of the external ear?

Describe the malformations of the auricle and the external auditory meatus.

What is a sebaceous cyst? its treatment?

What is the differential diagnosis between hæmatoma aura and perichondritis auriclæ?

Give the etiology, symptoms, and treatment of diffuse external otitis.

Give the etiology, symptoms, and treatment of circumscribed external otitis.

What are the usual causes of impacted cerumen, and how is it best removed?

Name the various forms of foreign bodies that may be found in the external auditory meatus.

Discuss the different methods of their removal.

Why should the physician give a guarded prognosis in cases where impacted cerumen or other foreign bodies have existed in the canal for some time?

CHAPTER IV.

DISEASES OF THE MIDDLE EAR.

Classification.—Considerable differences of opinion as to the proper anatomical subdivision of the middle ear and the classification of its diseases obtain among writers; but inasmuch as the inner surface of the membrana tympani, tympanum, mastoid process, and Eustachian tube is invested by a continuous lining mucous membrane, by means of which inflammatory conditions of one part usually extend to and involve those remaining to a greater or less degree, and as the condition of all exerts some influence upon the transmission of sound, it seems productive of lucidity to consider the affections of all these subdivisions under the above heading, as outlined in the opening chapter. Other questionable classifications will be indicated in the succeeding discussion.

INJURIES OF THE MEMBRANA TYMPANI.

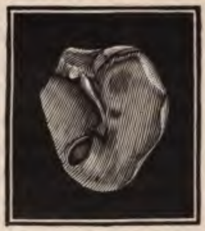
The drum membrane is frequently the recipient of injuries, which are **produced by** either a traumatic force or disturbance of the atmospheric pressure exerted upon the outer or inner surface of the membrane.

Lesions.—Traumatic lesions usually result from the accidental or intentional introduction of foreign bodies or substances into the external auditory meatus, when abrasions, ulcerations, perforation or rupture of the membrane may be produced. A bougie introduced into the tympanic cavity through › *Eustachian* tube may also accidentally injure the membrana tympani. The use of matches, tooth-picks, hair-pins, ear-spoons, and various other objects to scratch the canal or remove the cerumen, as employed by the laity, forms a prolific source of direct injury to the lining of the canal and the membrana tympani. The utmost precaution should be observed by the physician in the use of instruments within the meatus, as very distressing forms of lesion may result from an accidental manœuvre on the part of the patient or the operator while performing instrumental manipulations in the region of the drum membrane. The position, form, and extent of injuries in this structure are variable, depending upon the direction of the canal, the character of the penetrating object, whether it be pointed, blunt or sharp, rigid or flexible, smooth or roughened; and the force with which it is applied against the membrane.

Symptoms.—Coincident with instrumental wounding of the membrana tympani a loud, rumbling sound is heard, accompanied by a sharp, piercing pain. Nausea, vomiting, dizziness, tinnitus, and deafness, or an unconsciousness which terminates the agonizing scene, may ensue. After a time, depending upon the extent of the injury, the symptoms will abate; but with the development of a reactive inflammation a throbbing pain and harassing noise add to the discomfort of the sufferer. If a perforation or rupture has resulted, with an infection of the tympanum, a painful, suppurative condition of the middle ear may ensue and prolong the condition for several weeks, with a possible secondary involvement of the mastoid process. If the abrasion or perforation of the membrane be small, however, in the absence of infection resolution will occur within a few days, with little or no alteration of function; but with large, irregular ruptures, permanent openings through the drum membrane are liable to remain, which, together with the formation of cicatrices and adhesions within the tympanum, may impair the hearing markedly.

Rupture of the Membrana Tympani.—Fractures of the cranial bones which extend into the external auditory meatus, sudden condensation of the air upon either side of the drum membrane, and the hyperformation of fluids within the tympanum are additional factors in the production of ruptures of the membrana tympani (Fig. 64). The former may result from blows received upon the mastoid, temporal, or inferior maxillary bones, when, if the fractures involve the labyrinth or cranial cavity, a serous fluid may escape from the meatus, together with a copious hemorrhage, which indicates a most unfavorable prognosis for the restoration of function, if not for the patient's life. Suppuration may later add to the complexity of the condition. Ruptures resulting from a sudden change of air pressure may be caused by a slap or box on the auricle, impaction of a surf-wave, the proximate discharge of a cannon or gun, or the condensations and rarefactions incident to the inflation of the Eustachian tube, and the use of the pneumatic massage otoscope in the meatus. In the latter instances the accident is usually traceable to a weakened condition of the membrane, due to the presence of atrophic areas, calcareous deposits, or cicatrices. It is well, therefore, always to examine the membrana tympani carefully before employing the foregoing procedures, as the physician may thereby avoid an embarrassing accident. Perforation or rupture of the drum membrane, frequently attendant upon seropurulent inflammatory conditions of the tympanum, results from an imprisonment of abnormal secretion, which finally forces its way through the path of least resistance, *the inflamed* membrana tympani, to the external auditory

FIG. 64.



Rupture in the anterior inferior half of the membrane of a boy after a box on the ear. (Politzer.)

meatus. The appearance of a ruptured membrane resulting from noninflammatory causes will vary greatly, depending upon the method of production, its location and size, but in general they are situated in the antra-inferior quadrant, midway between the centre and periphery of the membrane, rarely extending the whole distance. In size it may present an elongated slit with the clean-cut edges slightly or markedly separated. Adhering near the edges of the wound may be noticed darkened reddish areas of coagulated blood. Through the gaping rupture the grayish-yellow mucous membrane of the normal tympanum may be seen, while running from the periphery inward a few congested bloodvessels of the membrane may be noted in the affected quadrant. Inflation of the Eustachian tube produces a characteristic blowing or whistling sound as the air escapes through the perforation or rupture of the drum membrane. The perforations incident to inflammatory conditions of the tympanum will receive further consideration under that subject.

It may not be amiss to add that injuries of the membrana tympani frequently present a medicolegal aspect, especially if a perforation or rupture has been sustained, when the physician may be called upon to testify as to the impairment of audition. In general, if there be no involvement of the tympanum from infection or the labyrinth from concussion, and the rupture be moderate in extent, the opening will close in the course of a few days with little or no disturbance of function. A moderate-sized permanent opening through the drum membrane does not of itself materially affect the capacity for hearing, although the laity usually consider it otherwise.

As regards the **treatment of perforated injuries**, it is best to allow Nature to take her course, as attempts to clean the meatus or apply antiseptic measures to the wounded membrane may be the means of carrying infection into the tympanum. For this reason, therefore, syringing of the canal or instillation of drops should not be employed. A pledget of absorbent cotton is loosely placed in the canal to protect the wound from external influences and take up any fluid that may be formed. If an inflammatory condition of the tympanum should ensue, its treatment would be that of an otitis media.

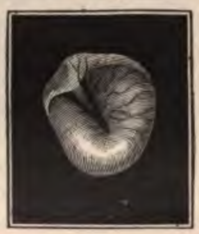
INFLAMMATION OF THE MEMBRANA TYMPANI.

Pathological alterations of the drum membrane occur either as a primary affection of the membrane or secondary to inflammatory conditions of the meatus and tympanic cavity, the latter being the more commonly observed form.

Synonym.—Myringitis.

Etiology.—The condition frequently appears as an idiopathic affection. It may be noticed coincident with a naso-pharyngitis; following an unusual exposure to a cold draft upon the ear; cold baths, douches, and sea-bathing are occasional causal factors; instillations of various irritating and caustic substances into the meatus are usually followed by a primary inflammation both of the membrana tympani and the lining meatal structures. Traumatic lesions frequently extend their inflammatory borders well into the surrounding membrane.

FIG. 65.



Hyperæmia of membrana tympani.

Pathology.—The first evidences of a myringitis (Fig. 65) are shown by a slight injection of minute vessels running from the periphery toward the centre of the membrane and coalescing with those which course along the region of the *long handle* of the malleus. As the inflammation develops, the hyperæmia becomes more marked until a diffuse or striated flush envelops the whole surface of the drum membrane and involves the contiguous dermal layer of the meatus. Coincident with the development of the hyperæmic condition a serous infiltration of the epithelial layer, accompanied by ecchymotic areas or the formation of clear, transparent vesicles, may be observed, which gives the membrane a dull, lustreless appearance, excepting where the pearly vesicles are situated. In the course of a few hours the vesicles disappear by absorption or rupture with a discharge of the serous contents, which may later become sanguineous in character, leaving a ragged excoriated surface behind. Abscess and ulcerations of the membrane sometimes result from the vesicles, though this is a rare complication.

Symptoms.—With a moderate inflammatory condition the symptoms are not specially marked; a varying amount of pain, sensation of fulness or pressure, some tinnitus, and little or no disturbance of hearing. In marked cases, however, especially if attended with the formation of vesicles or deep-seated abscesses, the pain becomes intense, particularly at night, and may continue with intermissions for several days. An ordinary case of myringitis usually terminates in resolution within the course of a few days, with complete cessation of all symptoms.

Diagnosis.—A primary myringitis can be confounded only with the secondary form, which results from an inflammatory condition of the tympanum, but the absence of marked pain, with little or no disturbance of audition would indicate a simple inflammatory condition of the membrane. In an involvement of the membrane resulting from an inflammatory condition of the middle ear, however, the hearing would be considerably impaired by the presence of a fluid transudate, which mechanically interferes with the transmission of sound.

Prognosis.—As previously indicated, the termination is usually favorable, resolution occurring within a few days.

Treatment.—As the symptoms are usually not marked and the course of the disease is limited, little or no interference with Nature's course is necessary; but it might be well to syringe the canal with a warm alkaline antiseptic solution to rid it of infection, and after drying insert loosely a pledget of cotton in the meatus. If the membrane becomes ulcerated, insufflations of boric acid may be made upon the surface. Abscesses or persistent vesicles may be emptied by paracentesis, care being taken that the instrument does not pass through the whole membrane, as an infection of the tympanum might result.

INFLAMMATION OF THE EUSTACHIAN TUBE.

Synonyms.—Eustachian tubal catarrh and salpingitis.

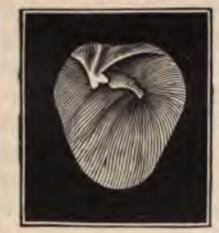
Etiology.—This affection of the Eustachian tube occurs as an acute or chronic disorder. The most frequent cause of a congestion or inflammation of the tubal lining membrane is an acute coryza or acute nasopharyngitis. In the exanthemata of childhood tubal obstruction results from the secondary involvement of the nasopharynx. Abnormalities of the nasal or pharyngeal cavities which interfere with proper respiration, such as adenoids in the vault, enlarged turbinates, septal deviations and spurs, hypertrophied follicular tonsils, irritating solutions or substances which may reach the Eustachian orifice, injuries from instrumentation, and impairment of the general health may be observed as causal factors in the production of tubal obstruction.

Pathology.—As the mucous membrane of the Eustachian tube is continuous with that of the tympanum and nasopharynx, inflammatory conditions of the latter frequently extend not only to the tubal lining membrane, but occasionally reach the tympanic cavity. Within the tube the mucous membrane may present simply a hyperæmic or injected condition, or a true inflammation may develop therefrom. In the latter instance the membrane becomes swollen and puffy, the lumen of the passage is markedly decreased or obliterated, transudation of a fluid adds to the flabby condition of the mucous membrane, and finally a thick, glairy, tenacious secretion completes the occlusion of the tube. As the cartilaginous portion of the canal is the more vascular, the above-mentiond process is usually confined to that region. With the closure of the Eustachian tube the atmospheric pressure within the tympanum is reduced through the rapid absorption of the contained air, which causes the membrana tympani and ossicular chain to be forced inward by the greater atmospheric pressure from without. If the tubal obstruction remains sufficiently long, the drum membrane will become so retracted as to rest in contact with the promontory of the inner tympanic wall. Later, a congestion of the tympanic vessels may occur with the formation of a serous transudation, which may completely fill the cavity and exert an outward pressure upon the membrana tympani.

Symptoms.—Coincident or following the development of an acute inflammation of the nasopharynx the patient may experience a sensation of fulness, numbness, or pressure in the meatus, which he may try to remove by inserting the finger into the canal and suddenly withdrawing it in such a manner as to create a vacuum. Occasionally sharp or dull intermittent pains may be noticed within the tympanum or pharynx. Tinnitus, high-pitched in character, is usually present. Impairment of audition, which may be very marked, mental and physical torpidity, a feeling of numbness about the aural region, sensations of dizziness, resonance of the patient's own voice, and a snapping sound while swallowing characterize the usual line of symptoms. Upon inspection of the membrana tympani it will be observed that the membrane is retracted or bulged inward to a greater or less extent, depending upon the duration and completeness of the tubal obstruction. The lower end of the malleal handle is drawn inward and backward (foreshortened), so that the antra-inferior surface of the drum membrane appears broader than usual. The short process of the malleus is particularly prominent, as are also the anterior and posterior folds of the membrana flaccida (Fig. 66). The cone of light is displaced, distorted, or absent. With inflation of the tympanum the retraction disappears (Fig. 67), and the function of audition is instantly restored, the sensation of fulness and numbness disappears, and the patient experiences a feeling of buoyancy but after a time the symptoms gradually return through the absorption of the air within the tympanum.

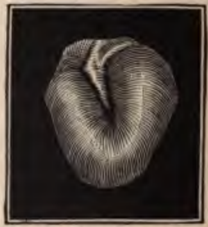
Diagnosis.—This condition is diagnosed by the presence of the foregoing characteristic symptoms and the complete restoration of hearing attendant upon inflation of the tympanum. If little or no improvement of audition follows the inflation, however, an inflammatory or catarrhal condition of the tympanum is indicated. Functional examination would show an elevation of the lower limit, together with a diminution in the duration of air conduction. If only one side were affected, or the two unequally impaired, Weber's test would be heard louder in the one more seriously involved.

FIG. 66.



Appearance of membrana tympani in a man aged thirty years. (Politzer.)

FIG. 67.



Condition of membrana tympani in same patient immediately after inflation.

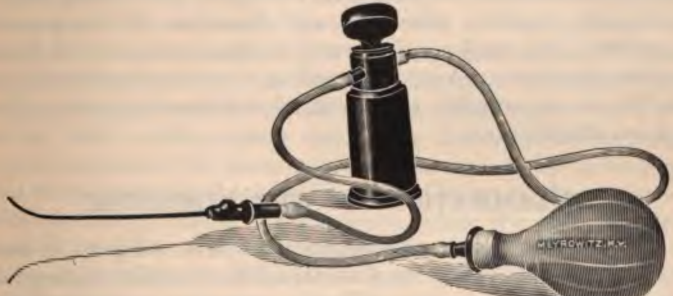
Prognosis.—An acute salpingitis dependent on an acute inflammatory condition of the nasopharynx will usually disappear spontaneously as the etiological affection improves; when the tubal obstruction becomes chronic, however, through frequently repeated attacks, the termination often is questionable.

Treatment.—Attention should be directed to the local tubal trouble, the causal conditions, and any predisposing systemic disorder, to effect a rapid and permanent resolution. To restore the hearing, quiet the harassing noises, remove the abnormal sensations about the ear, relieve the patient from his mental gloom, and quell the disturbances of equilibrium, repeated inflations of the tympanum offer a panacea. Either *Politzerization* or catheterization may be employed for this purpose. Occasionally a marked stenosis of the tube is encountered, which can not be overcome by either the air-bag or catheter, and is liable to become chronic. In this condition recourse may be made to the bougie—whalebone or celluloid —(Fig. 68), which is introduced into the orifice of the Eustachian tube through a catheter, and very gently forced upward through the canal until the obstruction is encountered and passed if possible. Great care should be exercised in this procedure lest the mucous membrane be injured, when a subsequent inflation might result in an emphysema of the adjacent tissue. In the chronic form of tubal stenosis graduated sizes of the instrument are employed until the stricture is sufficiently dilated to permit normal aëration of the tympanum. Stimulating vapors of alcohol, ether, chloroform, or tincture of benzoin may be applied to the lumen of the tube by means of the air-bag, or preferably the catheter, with vaporizer attached (Fig. 69).

FIG. 68.

Eustachian bougie.

FIG. 69.



Dench's vaporizer with catheter attached.

As to the treatment of the nasopharynx, this is always indicated if there be any pathological conditions present, either in the acute or chronic form, which may be instrumental in the production or prolongation of the Eustachian involvement. Free nasal respiration should be obtained by medicinal or surgical procedures, as may be indicated. In the acute forms of nasopharyngitis much can be accomplished by the judicious use of a warm alkaline antiseptic spray, followed by local applications of mild solutions of cocaine, or preferably adrenalin, to the swollen inferior turbinates, after which a bland antiseptic oily spray may be applied to the nasal cavity as a protective. The writer frequently swabs the pharyngeal orifice of the Eustachian tube with an adrenalin solution (1 : 1000), which is applied by means of an applicator, followed by nitrate of silver solutions (4 or 6 per cent.). In the chronic forms of salpingitis, the nose and pharynx are frequently the seat of pathological conditions which interfere with proper nasal respiration, thereby inducing a congestion of the tubal structures or mechanically obstructing its lumen. For the treatment of adenoids, enlarged turbinates, septal deviations, tonsillar affections, and disorders of the mucous membrane, the reader is referred to the discussion of these subjects elsewhere. Any predisposing systemic disorder should receive its appropriate treatment as indicated in works upon general therapeutics.

INFLAMMATIONS OF THE TYMPANUM.

Introductory to the consideration of this subject, it may simplify its conception to note that inflammatory diseases of the tympanic cavity occur either as **catarrhal** or **purulent disorders.** The former, which appear in an acute or chronic form, are characterized by a thickened, infiltrated condition of the mucous membrane, with a serous transudate or a hyperformation of its normal secretion, resulting from either a simple hyperæmia or true inflammatory involvement of the lining membrane; while in the latter conditions the reactive *phenomena* extend to the deeper structures, produce a more or less violent engorgement and swelling of the mucous layer, and terminate in the formation of a mucopurulent or purulent secretion, which may pursue either an acute or chronic course.

ACUTE CATARRHAL OTITIS MEDIA.

Synonyms.—Acute catarrhal inflammation of the middle ear; Otitis media catarrhalis acuta; Otitis media serosa.

Etiology.—Sudden changes in the atmospheric conditions, acute coryza, acute pharyngitis, influenza, the acute exanthemata, stenosis of the Eustachian tube, extension of infection to the tympanum from acute or chronic catarrhal conditions of the nasopharynx, adenoids in the vault, stenosis of the nasal cavity, introduction of fluids into the tympanum while gargling, bathing, employing the nasal douche, or violently blowing the nostrils, and general debility resulting from various systemic disorders, may singly or combined act as factors in the production of an acute involvement of the middle ear.

Pathology.—With the development of an acute tympanitis, the superficial structures become more or less injected or hyperæmic, accompanied by a swollen, infiltrated condition of the lining mucous membrane. A serous effusion, mixed with a hypersecretion of the mucous glands, soon fills the cavity with a thin, watery fluid, or a thick, turbid secretion which macerates the mucous membrane and holds in suspension the desquamated epithelial cells. Owing to the numerous folds or reduplications of the mucous lining, the process is frequently confined to the attic, while the remaining part of the cavity is unaffected. The inner layer of the membrana tympani becomes involved in the inflammatory condition of the tympanic space, which weakens its resistance through the process of maceration and desquamation. With an extension of the infiltration to the fibrous layer, the membrane becomes so devoid of strength as frequently to rupture under the excessive pressure of the tympanic secretions. The inflammatory process of the tympanum usually affects the lining membrane of the mastoid process, to a greater or less extent, through the intervening atrium. The Eustachian tubal structures also are generally simultaneously involved, which may proceed from the tympanum, but usually results from the same causality that produces the tympanitis.

Symptoms.—A few days after the development of an acute coryza or pharyngitis, the patient may notice a sensation of fulness in the Eustachian tube or the tympanum, followed by an impairment of hearing, tinnitus, and an occasional pain, which may be dull or piercing in character. As the disorder progresses, the symptoms increase in intensity, especially the involvement of audition and the sensation of pain. If the affection occurs in a child, the symptoms would seem to indicate the presence of a very grave condition, as they are more marked than those usually observed in the adult. Since the trouble frequently develops at night, the little sufferer appears restless, rolling the head from side to side and throwing the arms aimlessly about. Sometimes the hand will be applied to the affected region; but usually this is done in such a manner as not to attract the attention of the physician to the seat of the pain. A concomitant profuse perspiration, increased pulse-rate, and sudden rise of temperature, ranging from 102° to 104° F., give a clue to the severity of the affection; but the attendant is frequently none the wiser until a mucomucous discharge escapes from the external auditory meatus several hours later, to the astonishment of the unsuspecting observers. This is usually followed immediately by a cessation of all the symptoms. The patient becomes quiet, drops off to sleep, while the temperature gradually returns to or near the normal point. In the adult, however, owing to the greater resistance of the membrana tympani, perforation of the membrane does not occur as soon or often as with the youthful subject. If the retained secretions be not liberated from the atrium, they are liable to fill the attic and then extend to the pneumatic spaces of the mastoid process. When this occurs the patient experiences a sensation of fulness, pain, swelling, and tenderness in that region. Constitutional disturbances, more or less pronounced, usually make their appearance at this time. The patient feels exhausted, both mentally and physically, which is probably due in great measure to loss of sleep and rest.

The character of the discharge varies greatly, not only in different individuals, but in the same patient from time to time. It may be a clear, serous fluid, or a thick, turbid secretion, which may stop the opening in the drum membrane and produce a return of all the symptoms. An intermittent flow of the discharge is usually traceable to this cause, although it may be due to a reparative process in the perforated membrane. A sudden arrest of the discharge is usually indicative of an obstruction to its escape from the tympanum, and should always be regarded as an unfavorable symptom, which demands a careful investigation, as otherwise great damage may result. By an infection from the meatus the catarrhal form of otitis media may be changed to a purulent inflammatory condition, which is, indeed, an unfortunate occurrence.

Physical Examination.—With the advent of the affection the membrana tympani may first show a condition of retraction due to the stenosis of the Eustachian tube. The vessels about the long handle become injected and the hyperæmic area of this region gradually merges into the normal periphery. The short process stands out prominently, and the lower folds of the membrana flaccida generally present a greater degree of hyperæmia than the vibrating portion, traceable to the greater vascularity of the attic with its numerous folds of the mucous membrane and thick, spongy substructure of connective tissue. Later the whole surface of the drum membrane becomes reddened, the hyperæmia extending even to the lining of the meatus, being especially marked in the posterosuperior wall where the membrana tympani seems to merge into the meatal epithelial layer. As the fluid forms in the tympanic cavity, the drum membrane assumes an œdematous, lustreless condition and may begin to bulge outward, especially in the posterior portion, from the excessive pressure of the retained morbid secretions. The epithelium finally becomes so macerated that it is desquamated as a whitened débris. This process, together with a similar affection of the inner mucous layer, greatly lessens the resistance of the tympanic membrane, and thus favors the production of a rupture or perforation.

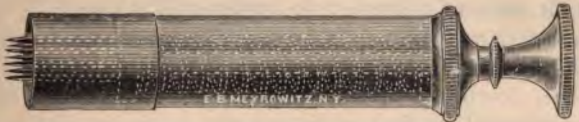
When the membrana tympani yields to the disruptive force of the tympanic fluid, a mucoserous discharge escapes from the meatus or forms a yellowish incrustation within the canal through evaporation of its watery constituents. If an inflation of the tympanum were now performed, the air would escape from the perforation with a hissing, bubbling sound, or if the secretions had all flowed from the tympanic cavity, the note would be sharp and whistling in character. The site of the perforation may be located by a small hemorrhagic area, which is usually differentiated with ease from the rest of the altered membrane.

Functional Tests.—As a rule the diagnosis is so evident and the necessity of treatment so urgent that the physician does not burden the patient in making these tests, aside from noting the degree of impairment in audition. This will be found to be more or less reduced; a vibrating tuning-fork placed upon the vertex of the skull is heard more distinctly on the affected side; air conduction appears markedly reduced; the lower tone limit is greatly elevated, while the upper is normal or but slightly reduced.

Diagnosis.—This is not difficult when the symptoms of fulness within the tympanum, rapid impairment of function, tinnitus, and aural pain are supplemented by the reddened condition of the membrana tympani, the existence of a horizontal line across its surface, which indicates the height of the retained secretions, a bulging in the posterior or superior portion, or the evidence of a perforation, attended by a mucoserous discharge within the meatus. The only condition with which this may be confounded is an acute purulent tympanitis, which can be differentiated only by the character of the discharge as it escapes into the meatus from a spontaneous or artificial rupture of the membrana tympani.

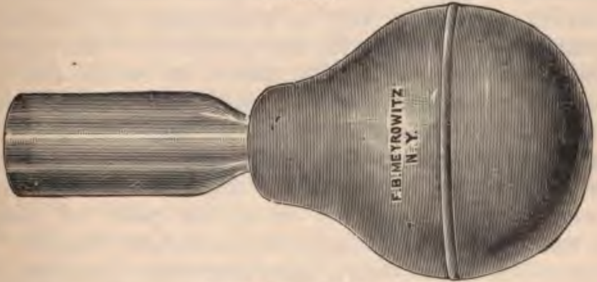
Prognosis.—The termination of a simple, uncomplicated, acute catarrhal process is generally favorable, especially with se who enjoy a healthful, hygienic mode of living, and when an early disappearance of the secretions is effected through absorption, spontaneous perforation, or incision of the membrane. When removal of the fluid is delayed, the process may become chronic through repeated attacks, and assume either a hypertrophic or proliferative condition. With the formation of cicatricial bands or a thickening of the mucous membrane, the mobility of the ossicles becomes restricted, with a resulting impairment of hearing, which is more commonly observed in the purulent type of involvement. Obstinate stenosis of the Eustachian tube, chronic nasopharyngitis, affection of the labyrinth, old age, the existence of systemic disorders attended by debility and anæmia, and the excessive use of alcohol are regarded as unfavorable conditions.

FIG. 70.



Bacon's scarificator.

FIG. 71.



Bacon's cupping-glass.

Treatment.—If the pain be particularly marked and constitutional symptoms be evident, the patient should be put to bed, a saline cathartic given, and an opiate administered to control the pain, although the physician should be careful that the drug does not conceal a grave condition, as the symptoms may be entirely masked thereby. The abstraction of blood by means of the natural, or preferably the artificial, leech and cupping (Figs. 70 and 71), applied in front of the tragus, is often abortive in the early stage. Dry heat in the form of a hot-water bag may also be used with benefit in lessening the pain. Should the nasal cavity be obstructed by a swollen condition of the mucous membrane, the stenosis may be relieved by first cleansing the passages with a warm alkaline spray, applying a cocaine or adrenalin solution to the parts by means of an applicator or spray, followed by a bland, oily nebula or spray, which would serve as a protective to the membrane. In case a pharyngitis be present, a mildly astringent gargle should be employed, or sprays may be used instead.

When nasopharyngeal inflammation is present, the Eustachian tube usually becomes involved from an extension of the process, evidenced by a retraction of the membrana tympani, which conditions may be ameliorated by inflation of the tympanum, together with the treatment of the nasopharynx indicated above. If after twelve to twenty-four hours the condition has not shown improvement, the canal should be syringed with a warm antiseptic solution (the writer preferring carbolic acid (1 : 40) on account of its anæsthetic properties), and an incision of the drum membrane made in the postra-inferior quadrant or at the point of bulging, which will frequently obtain in the membrana flaccida. The canal should be closed with a bit of absorbent cotton or a strip of gauze, which will serve as a protective and also take up the discharge. The secretion should be removed at least once daily by syringing; by use of the applicator, or instillations of warmed hydrogen peroxide; the meatus dried; boric acid insufflations made; and the cotton or gauze replaced. The cleansing of the parts should not be intrusted to a layman, as *infection* of the tympanic cavity is liable to occur thereby and add greatly to the seriousness of the condition. As resolution takes place, inflation of the tympanum should be practised to prevent retraction of the drum membrane and internal adhesions. So marked are the cessation of symptoms and improvement of audition following an incision of the membrana tympani that the writer is partial to this method of treatment. In the course of a week or so the discharge usually ceases, the injection of the membrana tympani disappears, and the function of hearing gradually returns to normal, providing no complications be present.

CHRONIC CATARRHAL OTITIS MEDIA.

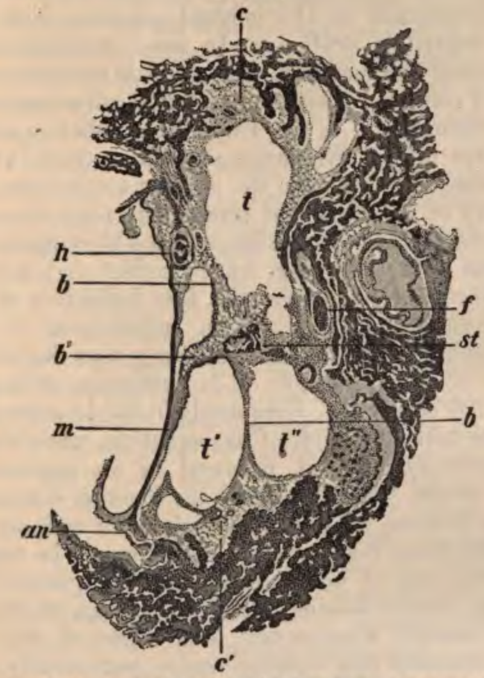
Synonyms.—Chronic catarrh of the middle ear; Chronic tympanitis; Otitis media catarrhalis chronica.

Etiology.—The causes of a chronic catarrhal involvement of the middle ear are similar to those which are productive of the acute form of otitis media, but a chronicity of the disorder usually obtains only with prolongation or frequent repetition of the etiological conditions. Chronic catarrhal affections of the nose or throat, stenosis of one or both nostrils by the presence of polypi, septal deviations, or hypertrophied turbinates, adenoids in the vault, enlarged faucial tonsils which may mechanically cause a stenosis of the Eustachian canal, the frequent taking of colds, and the deleterious effects of a changeable climate are the common causes of this affection. A delayed resolution of an acute catarrhal tympanitis predisposes to the chronic condition. An improper action of the levator or tensor palati muscles through paralysis or other causes may lead to involvement of the tympanum by improper ventilation. General disorders which impair the health, excessive use of alcoholic drinks or tobacco, and hereditary predisposition also play a rôle in the production of this catarrhal state.

Pathology.—Chronic catarrhal otitis media occurs in two forms, either as a hypertrophic (secretive) or hyperplastic (sclerotic, adhesive) catarrh of the middle ear. In **chronic hypertrophic otitis media,** which is the common type of involvement, the pathological alterations consist of a thickened vascular condition of the tympanic mucous membrane, which, in the early stage, is due to a venous congestion, but later to a true hypertrophy of the tissue elements. A fluid exudate, resulting from either a hypersecretion of the perverted mucous glands, a transudation from the bloodvessels of the engorged lining membrane, or a mixture of both, soon makes its appearance as a characteristic feature of this condition. The secretion may nearly fill the cavity and remain indefinitely or disappear periodically through absorption or a lessened activity of the exciting cause. The inner surface of the membrana tympani also assumes the pathological changes of the tympanic lining membrane, the middle or fibrous layer becomes thickened, and occasionally atrophic areas or deposits of calcareous matter occur therein. On account of a similar hypertrophic process in the mucous lining of the Eustachian tube the canal becomes chronically stenosed, the tympanic congestion increased, and the drum membrane markedly retracted, so that it frequently lies in contact with the promontory, to which it sometimes becomes adherent, although not so often as in the sclerotic form to be described shortly. With prolongation in the duration of retraction, the tendon of the tensor tympani frequently becomes shortened, so that inflation of the tympanum produces little or no change in the contour of the membrane. The presence of the tympanic secretion and thickened condition of the mucous membrane about the articulations of the ossicles and the intratympanic ligaments, together with the retraction of the drum membrane, so restrict the mobility of the ossicles that the function of audition is more or less impaired. Involvement of the labyrinth, which might occur through extension of the process to the cochlea, is fortunately a rare condition, although it sometimes appears in cases of prolonged severity.

In chronic hyperplastic otitis media the hypertrophic mucous membrane of the preceding condition is gradually replaced by an atrophic fibrous tissue, in which the secreting glands frequently are destroyed, thus constituting the so-called catarrh of the middle ear," or otosclerosis. Running through the exudative débris of a former hypertrophic state, connective-tissue bands are formed, which ramify in various directions across the tympanum and involve the ossicles (Fig. 72). The membrana tympani secondaria in the fenestra

FIG. 72.



Section through the tympanic cavity of a deaf man: *h*, handle of the malleus; *m*, tympanic membrane; *an*, annulus tendinosus; *st*, head of the stapes; *f*, facial nerve; *b*, *b′*, *b″*, newly formed connective-tissue bridges in the tympanic cavity: *t*, *t′*, *t″*, spaces in the tympanic cavity formed by these membranous bands. (Politzer.)

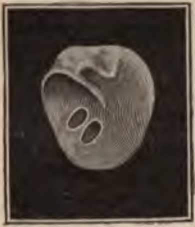
rotundum, the foot-plate of the stapes in the fenestra ovalis, and the tissues about the ossicular articulations become involved in the adhesive, sclerotic process, which frequently terminates in complete ankylosis through the deposition of calcareous matter, with serious impairment of audition. The fibrous bridges, adhesions at the joints, and sclerotic affection of the mucous folds and ligaments, especially in the attic, distort the position of the ossicles. So long as the foot-plate of the stapes be not immobilized, and the labyrinth be unaffected, the hearing may be preserved, but unfortunately these structures become sooner or later implicated in the general sclerosis, with a corresponding degree of deafness. In some instances the membrana tympani becomes thin, with marked atrophic areas, and is held in a retracted position by internal adhesions, especially to the promontory. As the Eustachian tubal mucous membrane shares in the tympanic alterations, with a resulting undue patency of its lumen, the tympanum is thus subjected to a liability of infection and traumatism from the use of the nasal douche, the act of sneezing, and the blowing of the nose.

Symptoms.—In both the hypertrophic and hyperplastic forms of chronic otitis media the first indication of involvement is usually evidenced by an impairment of audition, which sometimes comes on so insidiously that the unfortunate individual is often unaware of his affliction until a marked alteration has occurred. Frequently in the hypertrophic condition there will be periods of exacerbation, when the hearing will be seriously lowered, followed by an improvement as resolution occurs, but after a time the function does not return to normal because of the pathological alterations. In the hyperplastic form, however, the involvement is usually progressive, with few or no intermissions of improved audition. A harassing noise—hissing, blowing, ringing, or whistling in character—usually accompanies both forms of this disorder. In some instances this tinnitus occurs periodically, while in others the sounds are so incessant and distressing that the patient not only wearies of the constant din, but sometimes contemplates rest in suicide. These subjective sounds frequently disappear amid external noises, but return with renewed vigor with a cessation of the latter. It may occasionally be observed that the patient hears better when in a train, mill, or surrounded by other noises, than in a quiet ce. *This phenomenon* is usually attributed to an increased mobility of the ossicles, produced by the greater volume of sound, during which time the vibrations of lesser intensity overcome the rigidity of the ossicular chain, and are therefore more easily perceived. Sometimes this condition is noted in the hypertrophic form of involvement, but is usually characteristic of the sclerotic or hyperplastic type of otitis media. Pains of varying duration and intensity sooner or later add to the discomfort of the sufferer. In the hypertrophic condition the patient usually complains of a dull, drawing sensation in the tympanum, or a burning, neuralgic feeling in the region of the pharyngeal orifice of the Eustachian tube, especially if stenosis of the canal occurs; while in the sclerotic disorder the pain is periodical, sharp and stinging in character, of short duration, and occurs several times within the space of a day.

It is scarcely necessary to add that one side is usually first affected, although both may be involved at the same time. Impairment of hearing, tinnitus, and pain may occur simultaneously, while in other instances, only one or two of these symptoms may be present. An almost characteristic symptom of a marked hyperplastic otitis media is the sudden dulness of audition that occurs when a patient attempts to listen attentively. If the attention be not directed to a test of function, the hearing will be fairly good; but when an effort is made to perceive a conversation or particular sound, audition becomes impaired to such an extent that the patient can not hear sounds which were easily recognized a moment previously. This condition is probably due to an exhaustion of the auditory centres. So marked is the distress produced by the harassing noises and the lowered function of hearing that a general debility frequently results, with a perversion of mentality.

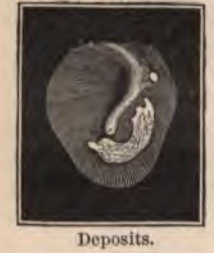
Physical Examination.—If the Eustachian tube be not markedly involved, the membrana tympani in both the hypertrophic and sclerotic types of otitis media will usually present a normal appearance; but more frequently a retraction of the membrane exists as a result of stenosis of the tubal lumen in the hypertrophic disorder, while in the hyperplastic condition the tube is generally unduly patent. A shortening of the tensor tympani muscle tends to hold the drum membrane in a retracted position, especially in the latter form of involvement. Atrophic areas (Fig. 73) and calcareous deposits (Fig. 74), together with adhesions of the membrana tympani to the promontory (Fig. 75), are frequently observed, especially in old cases. The use of the Seigel otoscope will aid in the differential diagnosis of these conditions. In some instances the membrana tympani presents a thickened, grayish appearance, while in others the structure is so uniformly thin and transparent that the incus-stapedial articulation may be seen, together with air-bubbles or fluids, within the tympanum.

Fig. 73.



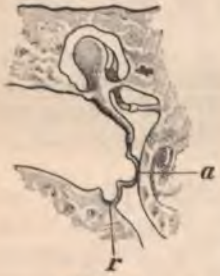
Atrophic areas.

Fig. 74.



Deposits.

Fig. 75.



Adhesion.

Functional Tests.—The acuity for hearing conversation is more or less impaired; tests with the acoumeter or watch usually show less involvement, although this order may be reversed occasionally. Patients frequently understand one person's conversation more distinctly than that of another. The lower tone limit is elevated, while the upper limit is usually normal or slightly altered. Duration of air conduction is shortened. Bone transmission is normal or increased to some extent, unless the labyrinth be affected, when both air and bone conduction would be shortened, and the upper tone limit correspondingly lowered. With the Rinné test, if the tuning-fork held upon the vertex be heard more distinctly in the worse ear, the middle ear alone is *affected; but,* if perceived more readily by the better ear an involvement of the labyrinth on the opposite side is indicated.

Diagnosis.—In uncomplicated cases of chronic catarrhal otitis media the diagnosis is usually not attended with difficulty, as is also true in the differentiation between the hypertrophic and hyperplastic conditions. A chronic impairment of audition, moderate tinnitus, dull pains, periods of improvement, stenosis of the Eustachian tube, a thickened condition of the membrana tympani with retraction, and amelioration of the conditions upon inflation of the tympanum are characteristic of the hypertrophic state; while a progressive impairment of hearing with no intermissions of improvement, a marked tinnitus, sharp, stinging pains in the tympanum, a thin, adherent condition of the drum membrane, patency of the tubal lumen with little or no improvement of audition upon inflation are indicative of a hyperplastic form of otitis media. These facts, together with the findings of the functional tests, clearly point to the location and character of the disorder.

Prognosis.—As a structural alteration of the mucous membrane is the chief pathologicnl condition in chronic catarrhal otitis media, the prognosis should always be guarded. If the labyrinth be unaffected, the function of audition not markedly lowered, and the condition has resulted from intranasal or pharyngeal conditions, which are amenable to medical or surgical procedures, the prognosis may be regarded as favorable; but with intratympanic adhesion, atrophic areas in the membrana tympani, a thin, flaccid condition of the drum head, undue patency of the Eustachian tube, marked retraction through a shortening of the tensor tympani, a progressive impairment of hearing, sharp, stinging pains, intense tinnitus, and a weakened physical condition, together with unhygienic surroundings, the prognosis becomes anything but favorable. Age is a very important factor to be considered in connection with the above data, as the younger the patient, the better are the chances for improvement.

Treatment.—As a chronic catarrhal involvement of the middle ear usually results from faulty nasopharyngeal conditions, it is imperative that any pathological alterations in this region should first receive the physician's attention. Free nasal respiration should be enjoyed at all times. Stenosis of the nasal passages frequently results from hypertrophy of the turbinal bones or a boggy condition of the overlying mucous membrane, together with the presence of septal deviations or spurs, and polypi or adenoids in the pharyngeal vault, which are best remedied by surgical procedures. Constitutional disorders, which may exert a harmful influence upon the aural condition, should receive appropriate treatment. In hypertrophic otitis media the Eustachian tube is usually stenosed by a thickened condition of its lining mucous membrane, which frequently disappears under proper treatment of the nasopharynx and tubal strictures, with a gradual improvement in the retraction of the membrana tympani and a betterment of audition. The thickened mucous membrane about the pharyngeal orifice may be reduced by cleansing the surface with a spray or cotton swab attached to an applicator which is curved like a catheter, and then making local applications of nitrate of silver (1 : 24) or argyrol (1 : 3). Should the stenosis persist, stimulating vapor of tincture of iodine, ether or chloroform in small quantities, ethyl iodide, or campho-menthol in alcohol (1 : 4), may be introduced into the Eustachian tube and tympanum by means of either the air-bag or catheter, the latter being more preferable. In most instances a judicious employment of the bougie will hasten resolution. Astringent solutions are sometimes injected into the tympanum through the Eustachian tube, or an artificial opening in the membrana tympani, for their effect upon a sluggish, thickened mucous membrane; but this procedure should be left to the specialist. Aural massage, which has for its object the securing of improved mobility of the ossicles, is usually not indicated in the hypertrophic state, especially when active, as an irritability of the condition may result therefrom. In the older cases attended with marked retraction and intratympanic adhesions massage may be tried; but even in these instances care should be exercised in its employment, as it may prove detrimental if used too long and frequently. Each case is a law unto itself in this respect; but so long as improvement in function follows its use, massage is indicated.

In the hyperplastic (sclerotic) form of otitis media, as would be inferred from its pathology, the physician faces a condition over which he has little or no permanent control, yet it is his duty to stay the process as much as possible. So long as the foot-plate of the stapes be not immobilized in the fenestra ovalis by adhesions or calcareous deposits, and the labyrinth be not affected, temporary improvement may be accomplished by aural massage and guarded forcible inflations of the tympanum, whereby adhesions may be lengthened or possibly separated and mobility of the ossicles improved. Often no betterment of audition follows this procedure, but a diminution of the harassing tinnitus may be accomplished, which is certainly appreciated by the sufferer. Care should be taken that the massage be not applied too vigorously or long, as the noises and impaired function may be aggravated thereby. Usually the process may be continued at each sitting until a flush begins to appear about the long handle of the malleus, when the treatment should be discontinued. This procedure may be used daily if carefully performed. So long as improvement is noticed in any respect, treatment should be continued; but if otherwise, it should be stopped. Stimulating inflations, as indicated in the hypertrophic state, may improve the vascularity of the tympanic mucous membrane, but are usually productive of no results. Removal of the ossicles, mobilization of the stapes, and tenotomy of the tensor tympani muscle, each has its advocates as a means of improving audition in selected cases of chronic catarrhal otitis media; but the results are still regarded as questionable. In the line of medicinal treatment sodium iodide (10 grains, t. i. d.), syr. hydriod. ac. (1 drachm, t. i. d.), and pilocarpine ($\frac{1}{8}$ grain, t. i. d., and gradually increased) are recommended for internal use as a means of promoting absorption of deposits in the tympanum and labyrinth.

[illegible] [illegible] [illegible] MEDIA

[illegible] suppu- [illegible] ear.

[illegible] middle ear [illegible] production [illegible] climatic [illegible] the [illegible] pharyn- [illegible] typhoid [illegible] frequent [illegible] may be [illegible] intro- [illegible] tym- [illegible] removal [illegible] to be [illegible] tympanum [illegible] ex- [illegible] of [illegible] and inda- [illegible] in [illegible] in-

[illegible]

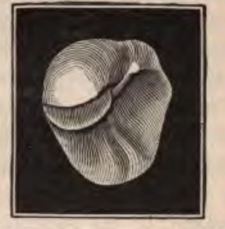
purulent secretion is very liable to be forced into the mastoid cells through the antrum, or secure vent through the membrana flaccida. As the tympanic cavity becomes filled with the purulent débris, the tension upon the swollen, macerated membrana tympani increases, until finally its weakened structure ruptures and the secretion escapes into the external auditory meatus.

Symptoms.—The symptoms may be the same as those mentioned under acute catarrhal otitis media, but usually the pain is of greater severity and the constitutional disturbances more marked. The symptoms are usually of greater intensity in the child than with the adult, and are more severe during the night, with a gradual improvement in the morning hours. Any physical or mental exertion aggravates the pain. The general weakness, apathy, and emaciated condition indicate the severity of the disorder. If the mastoid becomes involved, either before perforation of the membrana tympani occurs or subsequently, the local and constitutional disturbances become more intense. A sharp pain is referred to the mastoid region, tumefaction of the overlying tissues is observed, and a tenderness is elicited upon palpation of the part. Occasionally a paralysis of the facial nerve occurs from an extension of infection to its structure. Invasion of the meninges is indicated by an increased temperature, convulsions, delirium, and localized paralysis, if the brain be affected. Should the lateral or sigmoid sinuses become involved, symptoms of a pyæmic infection manifest themselves by a sudden rise of temperature, reaching 105° or 106° F. in a few hours and falling with equal rapidity to normal or even below that point, attended by profuse sweating and chills, which may recur at intervals of a few hours for some time.

Following a spontaneous or artificial rupture of the membrana tympani, a seropurulent or purulent discharge, which may be tinged with blood, escapes into the external auditory meatus. So rapid and complete is the cessation of all the symptoms following this phenomenon that the results seem indeed magical. The pain disappears, the feeling of tension subsides, the temperature gradually returns to normal, and the sufferer, exhausted both mentally and physically, drops into a refreshing sleep. In the course of a week or two the discharge generally subsides, when, if the perforation be not too extensive, the wound will close, leaving naught but a scar to indicate the involvement. Frequently, on account of a caries or necrosis of the ossicles, the tympanic wall, or the mastoid cells, the discharge continues for weeks, and may finally become chronic in character.

Physical Examination.—An inspection of the membrana tympani in the early stage usually reveals an injection of the membrana flaccida, which gradually changes to an engorgement and a bulging of the membrane outward, while the larger portion of the drum head below may show little or no alteration (Fig. 76). As the process advances, the whole membrana tympani assumes a dark-red, lustreless appearance. The outlines of the malleus disappear, the short process becomes indistinct, and the surface of the membrane appears moist from the extensive œdematous condition. As the secretion fills the atrium, a secondary bulging of the drum head may appear at any point, but is usually situated in the postra-superior quadrant, on account of the fact that the secretion trickles down the incus as it passes from the attic downward into the atrium. It is at this point, therefore, that a spontaneous perforation usually occurs; but if the secretion be retained mainly in the attic on account of an intense tumefaction of the mucous folds about the ossicles, the rupture will appear in the membrana flaccida.

FIG. 76.



Bulging of membrana flaccida.

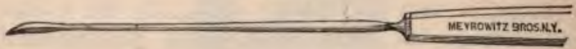
Functional Tests.—Again, as in the acute catarrhal otitis media, the condition is so evident and the treatment so urgent that these tests are not usually employed, but are identically those observed in the catarrhal affection as previously indicated. As the labyrinth is more liable to be involved in the purulent condition, either by extension through the fenestraovalis or rotundum, or the anastomosing vessels of the inner wall, this will be indicated by a marked lowering of the upper tone limit and a diminution in the duration of bone conduction.

Diagnosis.—As the signs and symptoms of an acute purulent inflammation are often similar to those of an acute catarrhal process, a differential diagnosis can not be established with certainty until the character of the discharge is ascertained as it escapes from a spontaneous or artificial perforation. The process may be catarrhal at the time of the perforation, and later become purulent, as evidenced by the discharge. In doubtful instances the presence of a perforation may be diagnosed by (1) ocular inspection, when, if the opening be sufficiently large, it may be readily seen; (2) the escape of a secretion through the drum head, if the perforation be not visible; (3) hearing the characteristic hissing or whistling sound as the air escapes from the perforation during an inflation of the tympanum through the Eustachian tube; and (4) passage of the air into the nasopharynx if a condensation of the air in the external auditory meatus be practiced.

Prognosis.—If the constitutional condition and the habits of the patient be good, the infection be of primary origin or has extended from an acute or chronic nasopharyngeal inflammation; a perforation of the drum head has occurred within a few days, which gives ample drainage to the tympanum; a subsequent rapid improvement in all the symptoms be noted, and there be an absence of mastoid involvement; the prognosis may be regarded generally as favorable for a complete restoration of function. On the other hand, infection resulting from scarlet fever, measles, or influenza is usually very severe, and the prognosis is frequently unfavorable. In fact, any complicating systemic disorder usually aggravates the local condition.

Treatment.—In the early stage vigorous treatment must be employed if the condition is to be improved. The one measure which promises the greatest satisfaction is the extraction of a large amount of blood from the region of the tragus by means of the natural or artificial leech. Dry heat may be applied to the external ear by use of a hot-water bag. A warm antiseptic solution (carbolic acid, 1 : 40) may be employed in syringing the external auditory meatus, both for its soothing effect upon the condition and as a means of cleansing the canal, previous to a perforation of the membrana tympani. The general condition of the patient should also receive appropriate attention. If the symptoms do not improve under this management, and especially if the drum head presents a bulging, an incision of the membrane should be made at the most prominent point, which is usually in the membrana flaccida or postra-inferior quadrant of the drum head. In operating at the former site care should be exercised lest the incus be injured or displaced. After an artificial or spontaneous perforation has been made, a moderate amount of blood escapes with the purulent secretion, which, if abundant, is regarded as favorable, as a depletion of the engorged structures is thus assured. The canal is now syringed with a warm antiseptic solution, and a strip of sterile gauze placed loosely in the passage for drainage and protection from external influences. As the packing becomes soaked with the secretion, it should be removed, the canal cleansed by means of the syringe or a cotton-tipped applicator, an insufflation of boric acid powder made upon the drum head for its antiseptic and absorbent properties, and a strip of gauze again inserted into the canal. If the discharge be abundant, the dressing should be replaced several times daily; but if moderate, once or twice a day will usually suffice. It is best that the treatment *of these cases* be not referred to the laity, as the condition

FIG. 77.



Knife for incision of drumhead.

may be aggravated by improper procedures. If the patient can not be seen as often as desired, after the usual dressings have been made, a pad composed of several thicknesses of sterile gauze may be placed over the meatus, where it may be held in position by means of bandages, which serve as a protection to the ear and take up the discharge as it escapes from the canal.

Sometimes the original perforation, either spontaneous or artificial, is not sufficiently large to afford proper drainage, especially if the discharge be copious or thickened in character. In this case the opening should be enlarged by an incision. Inflation of the tympanum and rarefaction of the air in the external auditory meatus are advocated as a means of removing the secretion from the tympanum. These procedures are not only usually painful, but often are useless, as with proper drainage the secretion escapes as readily as it is formed.

The treatment applicable to a chronic purulent condition, and in cases where the mastoid becomes involved, will be considered under the two following subjects.

CHRONIC PURULENT OTITIS MEDIA.

Synonyms.—Otitis media chronica suppurativa; Chronic suppurative tympanitis; Acute suppuration of the middle ear.

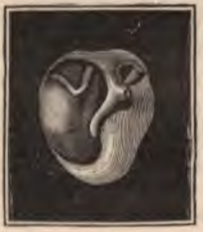
Etiology.—This disorder results from either a preceding acute catarrhal otitis media which has become infected or an acute purulent inflammation in which for various reasons resolution has failed to occur. An acute purulent condition which develops during an attack of scarlet fever, measles, influenza, tuberculosis, syphilis, or scrofula is always prone to become chronic in character. Unhygienic surroundings, insufficient and improper foods, and the abuse of alcohol aggravate the condition. Local conditions in the tympanum, together with diseases of the nasopharynx, sometimes also tend to the production of chronicity. Among these may be mentioned caries or necrosis of the ossicles, tympanic wall, or cells of the mastoid process; granulations and polypoid growths springing from the surface of the tympanic mucous membrane or edges of the perforation in the drum head; the retention of purulent débris within the tympanum or spaces of the mastoid and its subsequent caseation; purulent and eczematous conditions of the external auditory meatus by which infection is carried inward to the tympanum; and chronic catarrhal conditions of the nasopharynx, which keep the lining mucous membrane of the Eustachian tube in an irritable state, with an occasional extension to the tympanum.

Pathology.—As would be expected as a result of the chronic inflammation, the mucous membrane of the tympanum is hypertrophied and vascular. On account of the numerous folds of mucous membrane in the attic, its greater vascularity and thickness of its connective structure, the changes are more marked in this region. The lining membrane becomes so thickened from an infiltration of round cells, the formation of new bloodvessels, and the dilatation of the original vessels that the tympanic cavity is often greatly reduced in size. There may be areas in which the mucous membrane is atrophic, while other regions show not only the hypertrophic, vascular condition, but areas of granulation as well. Areas of bone necrosis may be present in different portions of the tympanic walls. The ossicles, especially the incus, may be partially or completely destroyed. The perforation in the membrana tympani is usually large and may be situated in any part of its structure. Sometimes the process is so extensive that the entire membrane, together with the ossicles, has been removed by the discharge. A secondary involvement of the mastoid is frequently observed and constitutes a grave condition, as the lateral sinus and meninges are liable to be affected sooner or later from this source, although an erosion of the thin upper wall of the attic and antrum may occur with equally fatal results. Extension of an infective process to the labyrinth rarely occurs, although the foot-plate of the stapes and the membrana tympani secondaria may be bathed in a purulent secretion for years.

Symptoms.—The only symptom of which the patient frequently complains is the presence of a discharge in the meatus. This may be so abundant that it escapes from the canal, or so scanty that only a scaly incrustation is formed in the meatus as a result of evaporation. Often the discharge will stop for a time and then appear again, following an attack of rhinitis or pharyngitis with an extension of the inflammation to the tympanum, or infection may occur from without. The hearing may be seriously affected or scarcely impaired, but this is not dependent upon the amount of discharge, as cases are frequently met where the function shows but little alteration, while the secretion is copious. Necrosis is usually indicated by a characteristically foul odor of the escaping discharge and the presence of granulations within the tympanum or their protrusion through a perforated drum head. As a result of uncleanliness granulations frequently appear from the edges of a perforation, and may be so extensive as to occlude the canal. Polypoid growths also frequently spring from the membrana tympani and vary greatly in size, usually approaching that of a small pea, although the writer has seen them not only fill the canal, but also protrude from the meatus like a miniature cauliflower. They are usually of a mucous formation, although fibroid polypi are sometimes seen. Involvement of the labyrinth to a slight degree is commonly observed in cases where the disease has existed for many years; but fortunately only the upper tone limit is altered, which does not materially affect audition for conversation. The symptoms which are characteristic of a sudden involvement are an acute dizziness, nausea, and loss of hearing. Paralysis of the facial nerve is sometimes observed; but this usually results from the primary acute condition. While constitutional disorders are usually one of the causes of the chronic purulent tympanitis, the general condition of the patient frequently suffers from the local disorder.

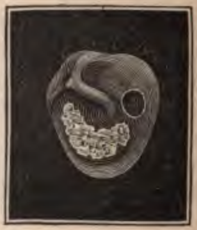
Physical Examination.—On inspection of the external auditory meatus a great variety of conditions may be presented. The discharge may be abundant or scant, purulent or mucopurulent, fluid or inspissated. The lining membrane of the canal may appear normal, eczematous, or irritable and excoriated from the constant presence of the discharge. The membrana tympani, which may present a normal or reddened color, will reveal one or more perforations. The opening may be so extensive as to involve the greater part of the membrane (Fig. 78), or so small that it is located with difficulty. The perforation may occur at any point in the drum head, but is more often observed in the posterior half. Calcareous deposits are also frequently seen (Fig. 79), which may also appear at any point. The ossicles, the chorda tympani nerve as it courses across the inner surface of the drum head below the short process, the promontory, the fenestra ovalis and rotundum, and granulations may often be seen in a case of large perforation. Granulation and polypoid growths have been mentioned previously. Necrosis of the internal wall or ossicles may be ascertained by a judicious employment of the probe, when a roughened, grating sensation will be imparted to the hand. Inflation of the tympanum produces a hissing or whistling sound as the air escapes from the perforation.

FIG. 78.



Large perforation.

FIG. 79.



Calcareous deposits.

Functional Tests.—The hearing may be normal or markedly lowered; the audition for conversation may be greater than that for the acoumeter or watch, or these conditions may be reversed; a tuning-fork placed upon the forehead or vertex is heard better in the affected or worse ear; the lower tone limit is raised, while the upper limit is normal if the labyrinth be unaffected; duration of air conduction is shortened, with a normality of bone conduction, although it may *eased*; and if the labyrinth has become affected, as it often is after many years, the upper tone limit is lowered and the bone transmission decreased.

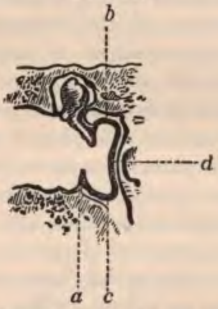
Diagnosis.—The history of the case and the findings of the physical and functional examinations leave no uncertainty as to the nature of the condition. Just when a case should be regarded as chronic has not been arbitrarily fixed; but after a discharge has continued for two or three months it is usually considered chronic in character. The discharge from a chronic eczematous condition of the canal or a diffuse external otitis may simulate that of a chronic purulent otitis media; but an inspection will reveal the absence of a perforation, and an inflation of the tympanum will not produce an escapement of air from the external auditory meatus.

Prognosis.—In making a complete prognosis it is necessary to decide as to the restoration of hearing, the duration of the discharge, and the risk to life incurred by the presence of a purulent, inflammatory condition within the tympanum. To answer these questions properly is often, indeed, a perplexing matter, but in general it may be stated that the functional alterations which have been produced after several months of duration will probably remain unchanged by treatment, although an improvement is often obtained. Progressive deafness is not so characteristic of this condition as is observed in the chronic catarrhal otitis media. As to the duration of the discharge, this will depend upon the presence or absence of a necrosis of the ossicles or the tympanic wall, drainage of the cavity, and involvement of the mastoid cells. With the securing of good drainage, removal or curettement of necrosing foci, and the elimination of mastoid complications, the prognosis becomes favorable. Regarding the risk to life which this condition presents, it may be stated that life insurance companies will not insure an individual who has a chronic purulent otitis media. As the lining mucous membrane serves as a periosteum to the underlying bone, a purulent affection of the membrane is liable to attack the bone, and thus extend to the intracranial structures by way of the attic or inner wall of the mastoid process, terminating in a meningitis, phlebitis, thrombosis, brain abscess, or pyæmia, from which recovery is doubtful.

Treatment.—So varied are the conditions that may be presented, that only a general outline of treatment can be given. A thorough cleanliness of the parts and the securing of perfect drainage are first to be considered. The discharge may be removed from the meatus by means of syringing, using a warm antiseptic solution, carbolic acid (1 : 40), bichloride of mercury (1 : 5000), or boric acid (saturated solution). Inflation of the tympanum or rarefaction of the air in the meatus may be employed to remove the discharge from the tympanic cavity. Should the perforation appear too small for proper drainage, the opening should be enlarged by an incision. If the discharge be abundant, the "wet treatment" should be employed after the secretion has been removed by syringing and the canal and drum head have been dried by means of a cotton-tipped applicator. This consists in the instillation of antiseptic or astringent solutions—*e. g.*, nitrate of silver (1 : 48), argyrol (1 : 4), or boric-alcohol (1 : 24), after which a strip of sterile gauze or cotton is inserted into the meatus. In some cases it may be necessary to change the dressing several times daily, but usually once or twice a day will suffice. If polypi or large granulations be present, these should be removed by means of forceps, curette, or snare. Smaller granulations usually disappear after the use of the boric-alcohol solution. If the discharge be scanty, the "dry treatment" is indicated. The secretion is removed by means of a cotton-tipped applicator, and then an insufflation of boric acid, alum and boric acid (equal parts), aristol, or nosophen is made into the tympanum through the perforation, care being exercised that the powder be not sufficient to interfere with a proper drainage. A piece of gauze or cotton inserted into the canal completes the dressing. Sometimes it may be necessary to alternate the wet and dry treatments. After employing both forms of treatment for several weeks, if there be little or no improvement, necrosis is indicated, especially when granulations are present in the tympanum and a characteristically foul odor remains in spite of thorough cleanliness. Instillations of iodoform-alcohol (saturated solution) are recommended in this condition; but if these prove ineffectual, the advisability of removing the ossicles, especially the incus, or curettement of the tympanum is to be considered. As the mastoid cells also are frequently involved, it may be necessary to clean out both the tympanum and mastoid before a cessation of the discharge is obtained.

Adhesion of the perforated membrana tympani to the promontory is frequently seen (Fig. 80), which fact often accounts for a marked impairment of audition, as the mobility of the membrane and ossicles is thus restricted; but so long as the hearing is fairly good, it is best not to attempt their separation, as the condition might be aggravated. Moderate and large-sized perforations usually remain patent after the discharge has ceased, exposing the tympanum to reinfection at any time, while the smaller openings frequently close spontaneously, or may be aided in this process by irritation of the edges by means of nitrate of silver solutions or a curettement. Where a moderate or even large perforation persists, the hearing often can be improved by the aid of an artificial drum membrane, which is placed in contact with the membrana tympani, being usually composed of a disk of rubber, to which a slender shaft is attached as a means of introduction and removal. A disk of paper or absorbent cotton may be used instead.

FIG. 80.



Adhesion of drumhead to promontory.

INFLAMMATION OF THE MASTOID.

Synonyms.—Acute or chronic mastoiditis.

Etiology.—Involvement of the mastoid cells occurs as a primary or secondary disease. The former condition is rare, resulting from traumatism, exposure to cold and dampness, or specific diseases, as syphilis or tuberculosis. The latter, which may be catarrhal or purulent, results from an extension of a tympanic inflammation through the antrum, as a rule. Infection may also occur through the postra-superior meatal wall from inflammatory conditions within the external auditory canal. When the anatomy of the tympanomastoid is considered, it is a wonder that serious involvement of the mastoid cells does not occur more frequently, especially when suppurative inflammation of the tympanum is of long duration. Nevertheless a mastoiditis is liable to develop at any time and endanger the life of the sufferer.

Pathology.—With every inflammatory condition of the tympanum there is more or less congestion of the lining mucous membrane of the mastoid cells, which, together with the fact that a secretion in the tympanum tends to flow through the antrum into the mastoid cells when the patient lies upon his back, accounts for the presence of pus in the mastoid of every acute purulent otitis media, as has been noted by Politzer. So long as does not obtain an actual inflammation of the mastoid lining membrane no symptoms are evident. As a result of the chronic congestion of the mastoid mucous membrane, accompanying a chronic purulent otitis media hypertrophy of the tissues lining the mastoid cells occurs, with an infiltration or deposition of calcareous matter, which often obliterates the pneumatic spaces, transforming the mastoid process into a solid bony structure. If the acute congestion or chronic hypertrophy of the membrane lining the air-cells be sufficient to close the openings whereby the cells communicate with the antrum and infective material is thereby encased, an abscess may develop, with necrosis of its enclosing walls. In some instances the communicating openings between the mastoid cells and antrum are large, while in others they are very small, so that a congestion, hypertrophy, or inflammatory œdematous condition of the lining membrane, which in one case would result in mastoid involvement, in another would prove insufficient. As a result of a chronic suppurative mastoiditis, necrosis of the bony walls between the air-spaces often occurs to such an extent as to obliterate them entirely, converting the spaces into one large cavity, which may be filled with granulation-tissue. The débris of necrosis escapes from the tympanum as a purulent discharge or may remain within the mastoid as a caseated mass.

Symptoms.—If the disease be primary, the patient will complain of a feeling of fulness or tension within the mastoid, accompanied by a dull constant pain behind the auricle, which is more severe during the night. Within the course of a day symptoms of a tympanic involvement may appear, due to extension of the process to the tympanum. Constitutional disturbances are not marked, the pulse being a little accelerated and the temperature raised but a degree or so. If the tip of the mastoid be involved, movements of the head may increase the pain in the mastoid, due to the traction of the sternocleidomastoid muscle upon the process. Œdema of the overlying tissues frequently occurs. When involvement of the mastoid is secondary, the symptoms are identically those cited above, with the exception of a previous tympanic disorder from which infection of the mastoid has occurred. If drainage of the mastoid abscess through the tympanum has not been obtained or be inadequate, the discharge will be evacuated from the mastoid at some other point of less resistance. This may occur through the external cortex or from the tip, when a fluctuating swelling will develop at the point of exit, or through the supra-posterior wall of the external auditory meatus, when the discharge will escape into the canal. Involvement of the intracranial structures may occur through the intervening bloodvessels or from an evacuation of the discharge through the inner wall of the mastoid. With the development of meningitis the patient complains of a headache, which may at first be intermittent, but later becomes constant and more severe, attended with photophobia, restlessness, insomnia, nausea and vomiting, and a constantly high temperature. A characteristic symptom is the rigidity of the neck. Paralysis of the third and sixth nerves frequently intervenes, with a resulting dilatation of the pupil and strabismus. With a localized meningitis or extradural abscess the symptoms are less severe and localized, while the rigidity of the neck, photophobia, and vomiting are absent. Involvement of the lateral sinus, phlebitis, is indicated by a sudden rise of temperature, when 104° or 105° F. is frequently indicated, followed by a rapid fall to the normal or subnormal point, attended by profuse sweating and chills, which may recur at several intervals during a day. As the involvement becomes more extensive, symptoms of a pyæmic condition become more pronounced. With the development of a thrombus disturbances in the circulation occur, evidenced by an œdema of the orbital and nasal tissues. As the thrombus extends downward into the internal jugular vein a deep tenderness, together with a swelling of the surrounding structures, develops. If emboli be formed, evidences of a metastatic infection occur, which in the lungs usually results in a septic pneumonia. With the development of a brain abscess the symptoms are indefinite, especially in the early stages; but when observed in connection with the existence of an acute or chronic purulent tympanitis or mastoiditis, the presence of intracranial pressure-symptoms, a pyæmic condition, and evidences of irritation or destruction of certain areas in the brain would indicate an involvement of this organ. With a chronic abscess, however, the symptoms are usually latent, with few or no positive indications of its existence, until a hemorrhage from an eroded bloodvessel or rupture of the encapsulated abscess suddenly renders the patient comatosed, accompanied by symptoms of paralysis. The acute form of brain abscess runs its course within a few days or weeks, depending upon its location and development, while a chronic abscess may exist for months or even years if encapsulated, as it usually is.

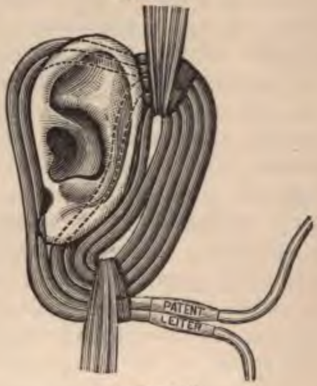
Physical Examination.—If the mastoiditis be primary, an inspection of the membrana tympani may later show evidences of an acute catarrhal or purulent tympanitis; while if secondary the latter conditions would be present before the mastoid involvement occurred. There may exist a swelling of the overlying mastoid tissues or areas of fluctuation; but the most characteristic signs of a mastoiditis are a tenderness of the region, elicited by firm palpation as compared with the opposite side, and an injection, together with a bulging of the postra-superior wall of the external auditory meatus near the annulus tympanicus.

Diagnosis.—When taken into consideration with the existence of an acute or chronic purulent otitis media, the development of a dull or sharp pain in the mastoid region, which increases in severity at night, evidences of a constitutional disturbance, a rigidity of the neck muscles, œdema of the tissues covering the mastoid bone, a localized tenderness about the antrum or tip, and a bulging of the postra-superior wall at its inner extremity, considered as a whole, these present a characteristic picture of a mastoid involvement. A differential diagnosis should consider inflammatory conditions of the external auditory meatus and tympanum.

Prognosis.—Inflammation of the mastoid process should always be regarded as a grave condition because of its liability of extension to the intracranial structures; but if recognized early and appropriate treatment be applied the prognosis is usually favorable so far as life is concerned. Following a chronic purulent otitis media, especially when the perforation of the membrana tympani occurs in the membrana flaccida, which indicates a marked involvement of the attic, intercurrent constitutional disorders, delayed and improper treatment, and the weakened resistance of old age are unfavorable conditions.

Treatment.—If seen at an early date, the condition may be aborted by anti-inflammatory measures. If an examination of the membrana tympani presents evidences of a bulging or intratympanic secretion, an extensive incision of the drum head should be made. If an original spontaneous or artificial perforation seems inadequate, it should be enlarged to secure proper drainage. The natural or artificial leech may be applied with benefit over the region of marked tenderness. Cold applications are of great value, and may be applied by means of an ice-bag, or preferably with the Leiter coil (Fig. 81), which may be molded to fit the mastoid perfectly. The application should be constantly employed for a day or so, when, if a marked improvement, especially in the local tenderness, has not occurred, an operation is indicated. A semicircular incision (Wilde's) through the overlying mastoid tissues, made one-half inch from the attachment of the auricle, is highly recommended by some writers as a means of local depletion and a first step toward a radical operation if necessary. If the inflammatory condition fails to improve steadily under the abortive treatment, the mastoid should be opened and its contents completely removed in the following manner: The field of operation, instruments, accessories, and hands of the operator having been prepared for an aseptic operation, a semicircular incision of the soft parts is carried from a point about one-half inch above the attachment of the auricle, backward and downward, keeping parallel to the auricular attachment and terminating at the tip of the mastoid. The periosteum is now elevated or dissected from the bone and the

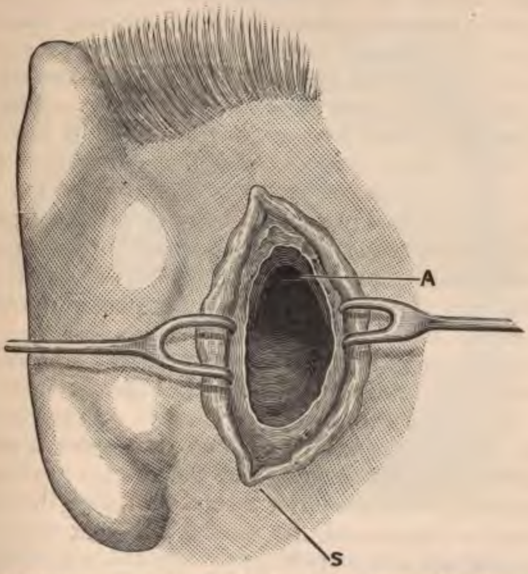
Fig. 81.



Leiter coil.

osseous structure thoroughly exposed by means of retractors, which are held by an assistant, the auricle being pulled forward so as to lie upon the side of the head (Fig. 82). The hemorrhage is controlled by the use of hot sponges and artery forceps. The surface of the mastoid bone is thoroughly examined for areas of necrosis or the existence of a fistulous opening, especially if a fluctuating swelling obtains previous to the operation. If these exist, the openings are enlarged by means of a gouge or a chisel and mallet, and followed inward to their origin. Should the surface present a healthy appearance, the primary opening of the mastoid is made into the antrum by means of the chisel, the point of entrance being effected just below the line of the superior wall of the meatus

FIG. 82.

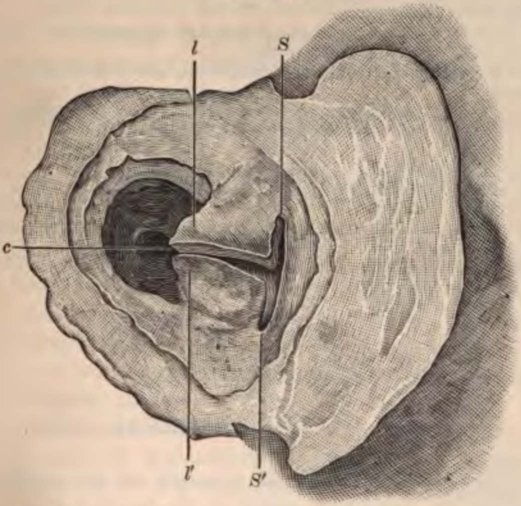


Mastoid process opened: *A*, antrum; *S*, tip of mastoid. (Politzer.)

and about one-quarter of an inch backward from the posterior wall or anterior edge of the mastoid bone. When the antrum has been exposed, the cortex of the mastoid is chiselled away from this point downward toward the tip until a sufficient amount has been removed to expose thoroughly all parts of the mastoid process. The cells are now all broken down, and every vestige of a necrotic or granulating area is completely eradicated. A free communication of the antrum with the tympanum should be established, which may be proved by syringing an antiseptic solution into the antrum, when it will escape from the external auditory meatus through a previous perforation of the drum head. The cavity of the mastoid is packed with sterile gauze, and the flaps of overlying tissue allowed to regain their former position, when a gap remains between their edges, through which the dressings may be changed. A thick pad of gauze is placed over the field of operation, and retained in position by means of bandages about the head. The dressings are changed daily or less frequently as indicated, until the cavity becomes perfectly healthy, when the gaping wound may be closed by a plastic operation if a neat cosmetic result be desired.

In the more complicated forms of mastoiditis especially, if the process be chronic, with a marked involvement of the antrum or tympanum, a more extensive operation will be necessary than that indicated above. In some instances it will be advisable after opening into the antrum to chisel away the intervening supra-posterior meatal wall, thus throwing the antrum and external auditory meatus into one cavity; while if the tympanum also be sufficiently affected, the whole intervening bony structure, together with the membrana tympani and ossicles, should be removed, thus converting the tympanum, antrum, and external auditory canal into a common cavity. As an extensive denuded osseous surface results from this procedure, some form of plastic operation must be made to dermatize the cavity. This is accomplished by a division of the postra-superior meatal lining integument (Fig. 83), whereby one or more flaps are formed. After the auricle is placed in its normal position, the flaps of skin are pressed against the posterior and superior surfaces of the cavity, and retained in position by tampons of gauze, which are changed as occasion demands. The flaps of integument gradually grow toward each other until the whole cavity thus becomes dermatized. Whether the wound behind the auricle should be partially or completely closed at the time of the operation will depend upon general surgical conditions, but it is better to err upon having a sufficiency of drainage than to incur the risk of retained secretions.

FIG. 83.



l, Longitudinal incision through the posterior wall of the membranous meatus; *S*, *S'*, superior and inferior incisions which are made at right angles to *c*, when the concha passes into the auditory meatus; *l*, *l*,' superior and inferior flaps which are made from the posterior wall of the meatus. (Politzer).

The danger of a mastoid operation lies in a possibility of injuring the lateral sinus or facial nerve, and the accidental entrance of the cranial cavity, whereby infection of the brain or its meninges may result, though fortunately this is a rare complication. The treatment of intercranial involvements resulting from an aural affection consists in remedying the primary condition, together with such specific or general treatment as would be indicated in a similar affection uncomplicated by an aural disturbance.

QUESTIONS.

What are the different ways whereby the membrana tympani may be injured?

How is a myringitis differentiated from the appearance of the drum head in acute otitis media?

What are the signs and symptoms of a stenosis of the Eustachian tube? Its pathology and treatment?

Give the etiology, pathology, signs and symptoms, diagnosis, prognosis, and treatment of acute catarrhal tympanitis.

What are the pathology, differential diagnosis, and treatment of acute purulent otitis media?

Give the etiology, pathology, differential diagnosis, prognosis, and treatment of the hypertrophic and hyperplastic forms of chronic catarrhal otitis media.

What are the dangers of a chronic purulent otitis media?

Give the pathology, symptoms, and treatment of an acute inflammation of the mastoid.

What are the signs and symptoms of a meningitis resulting from an aural infection?

What are the dangers of a phlebitis of the lateral sinus? Its treatment?

CHAPTER V.

DISEASES OF THE INTERNAL EAR.

NOTWITHSTANDING the vast amount of scientific investigation which has been expended in the study of the diseases of the labyrinth, our present knowledge of the pathology and significance of certain symptoms is still very incomplete. This fact is, of course, due to the inaccessibility of the internal ear to physical examinations during life, and the infrequency of postmortem opportunities whereby clinical manifestations of aural involvements may be explained. In spite of these limitations, however, some of the less obscure conditions are fairly well understood.

In considering the disturbances of audition referable to an *affection* of the labyrinth, it should be remembered that patho*logical alterations* which interfere with the function of the internal ear are not always located in the labyrinth itself, but may be traceable to the presence of a lesion in the auditory nerve or its cortical centre in the brain, which interferes as effectually with the power of hearing as though the disorder were located in the labyrinth alone. It is, therefore, well to regard the auditory tract as part of the perceptive mechanism of hearing. Anæmic, hyperæmic, hemorrhagic, inflammatory, hypertrophic, and atrophic alterations are the conditions which usually account for an involvement of the labyrinthine function. Although a primary invasion of the internal ear sometimes occurs, the disturbances usually appear secondary to a local or systemic disorder.

On account of the intimate vascular communication of the tympanum with the labyrinth through their intervening wall affections of the former sometimes extend to the labyrinthine structures. When associated with an acute catarrhal or purulent otitis media, the pathological alterations may consist of a hyperæmia, infiltration, ecchymosis, or actual inflammation, with a resulting disturbance of the intralabyrinthine pressure. As previously indicated, a sclerotic affection of the vestibule and cochlea frequently develops in the course of a chronic hyperplastic otitis media, but rarely occurs in connection with a hypertrophic process. With the oncoming of old age a physiological impairment of perception occurs as a result of senile changes in the auditory nerve and its terminations in the cochlea. Sclerotic changes in the membranous labyrinth sometimes appear also, which terminate in an ankylosis of the stapes.

Owing to its connection with the intracranial structures, inflammatory conditions of the brain and its investments frequently produce a temporary or permanent impairment of the auditory tract. Most productive of harm is an epidemic cerebrospinal meningitis, which is so frequently observed in childhood. Hydrocephalus, cerebral tumors, acute or chronic cerebral abscesses, acute or chronic inflammations of the brain, and disorders of the cerebellum and spinal cord may also be mentioned as etiological factors in the production of deafness.

The acute exanthematous diseases are very productive of impairment in the perceptive mechanism, as well as in the tympanum, especially scarlet fever, measles, influenza, and diphtheria, which frequently cause a hyperæmic, ecchymotic, or œdematous disorder of the auditory nerve or its labyrinthine terminations, either through a toxicity of the blood or secondary inflammatory conditions. Because of the susceptibility of the auditory tract, and the frequency of acute infectious diseases during childhood, deafness, resulting from an involvement of the perceptive tympanic, as well as the structures, develops more often during this period of life.

The following influences may also act as etiological factors in the production of labyrinthine changes: specific constitutional disorders (tuberculosis, syphilis, and uric acid diathesis), heredity (often overestimated), certain drugs in the system (quinine, salicylic acid, and alcohol), metallic poisons used in the arts (lead, mercury, arsenic, phosphorus, and carbon disulphide), concussions of the labyrinth resulting from intense sounds and sudden changes in atmospheric pressure (report of cannon, explosions, escaping steam, and the effects of a blow upon the auricle, forcible inflation of the tympanum, and sudden rarefaction of the air in the meatus), disturbances of circulation (thrombosis, embolism), and affections of the mind whereby vasomotor changes occur.

Symptoms.—In general it may be stated that disorders of the perceptive mechanism manifest themselves by symptoms of irritation or paralysis. In the former conditions the patient complains of hyperæsthesia of sounds, tinnitus, vertigo, nausea, and vomiting, while with the latter dulness of hearing, qualitative changes in perception, or deafness are noted. As irritative conditions frequently terminate in a paralytic disorder, a combination of symptoms occurs, which may be elucidated or rendered more complex by the presence of an incidental or causal disorder which may be local or systemic in character.

Diagnosis.—Owing to the vagueness and complexity of the symptoms presented in an involvement of the internal ear, it is necessary to resort to a systematic functional test as a reliable means of diagnosis; but even then the difficulties are not often solved. In an affection of the auditory tract it is frequently impossible to decide whether the lesion be located in the labyrinthine terminations, auditory nerve, or its cortical centre in the brain. A simultaneous disorder of the tympanum and labyrinth may exist, when it becomes a task to determine which condition accounts for impairment of audition. Quantitative tests (acoumeter, watch, or voice) throw no light upon the subject, as the extent of impairment only is indicated thereby. By means of the qualitative tests, however, as previously noted (page 32), the physician can differentiate between affections of the conducting and perceptive mechanisms.

Prognosis.—This will depend upon the cause, extent, and duration of the affection. If the disorder be of recent date, the impairment of hearing not marked, and the causal condition can be remedied or improved, the prognosis is favorable for restoration of function. Chronic impairment with a marked loss of hearing offers an unfavorable termination, although a most aggravated condition may improve to a satisfactory extent. Involvements of the perceptive mechanism occurring in the course of epidemic cerebrospinal meningitis, scarlet fever, and measles are usually regarded as serious, so far as impairment of audition is concerned.

Treatment.—Attention should first be directed to an improvement or removal of the etiological condition. In the case of anæmia of the labyrinth due to a systemic disorder, an abundance of nourishing food, systematic out-of-door exercises, and the use of iron and arsenic preparations internally will usually result favorably. Hyperæmic conditions require a depletion of the system, which may be accomplished by the administration of salines. Leeches may be applied locally, followed by cold compresses. The diet should be simple, the habits regular, and the use of stimulants avoided. Hemorrhagic and inflammatory affections may be treated in a similar manner. Iodides may be given for their absorptive effect. If the tinnitus be marked, large doses of sodium bromide may be used with benefit.

QUESTIONS.

Why is our knowledge of the pathology of disorders of the perceptive mechanism so limited?

What are the common affections of the labyrinth?

Give the etiology of diseases of the internal ear.

How do disorders of the perceptive mechanism manifest themselves symptomatically?

Why will a lesion in the auditory centre of the brain produce deafness as effectually as though it were located in the labyrinth?

How are affections of the tympanum and labyrinth differentiated from each other?

What is the prognosis of labyrinthine disorders?

Give in a general way the treatment of diseases of the internal ear.

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